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RESPIRATORY CARE

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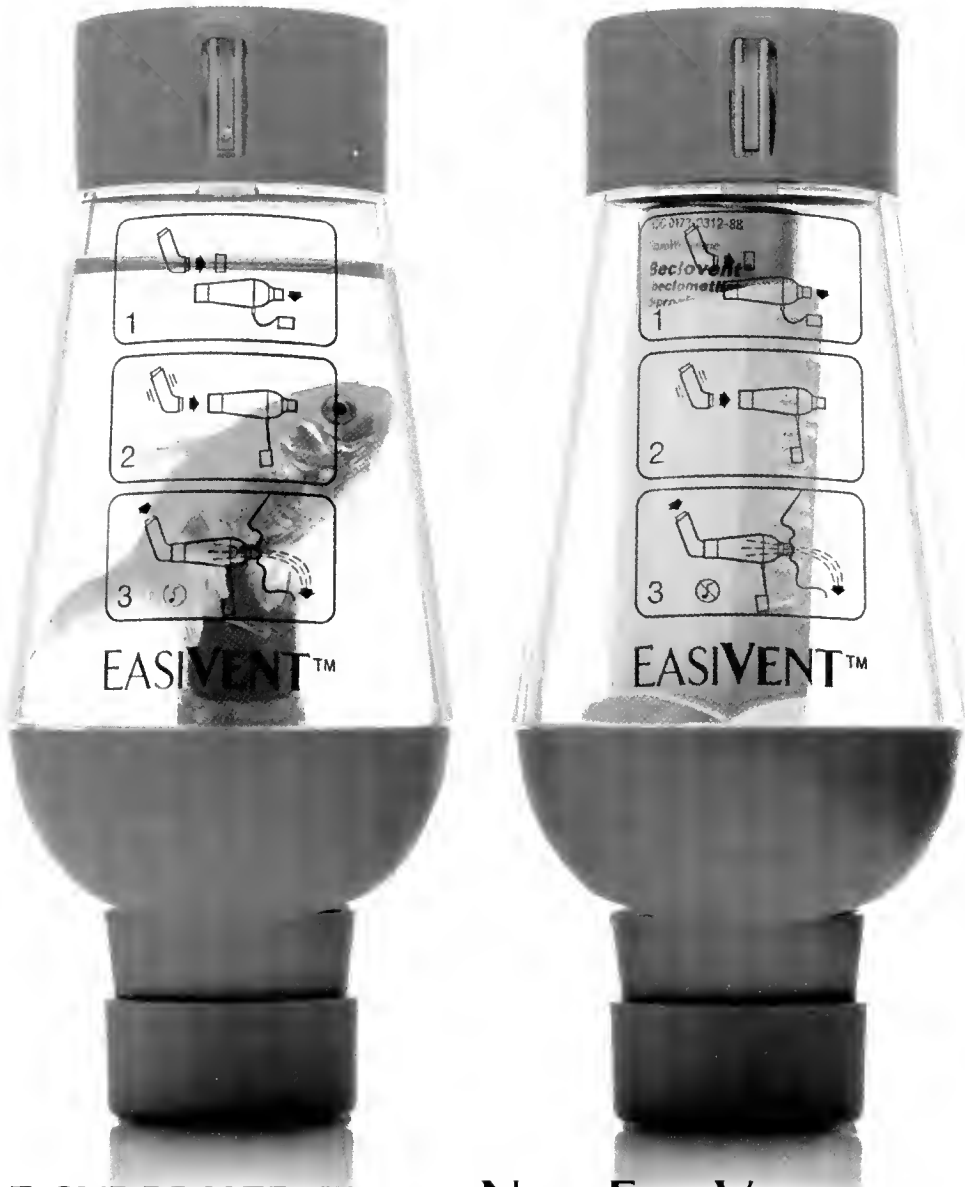
44TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE

THORACIC IMAGING IN THE INTENSIVE CARE UNIT

- Editorial: Improving Clinical Skills and Access Means Better Patient Care
- Pitfalls in Portable Chest Radiology
- Chest Imaging in the Cardiac ICU
- Chest Imaging in the Trauma ICU
- Chest Imaging in the Neurosciences ICU
- Chest Imaging in the Surgical ICU
- Chest Imaging in the Neonatal and Pediatric ICU
- Uses of Thoracic Computed Tomography in the ICU



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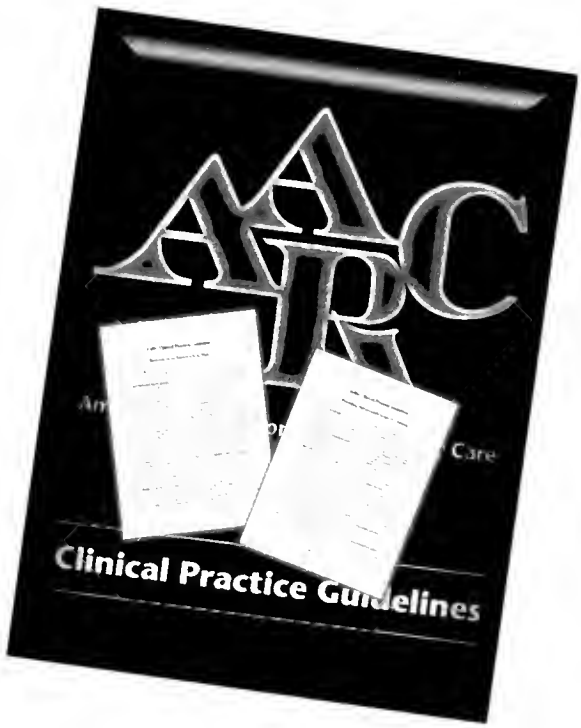
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SPECIAL ISSUE

THORACIC IMAGING IN THE ICU

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Eric J Stern MD
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EDITORIALS

Thoracic Imaging in the Intensive Care Unit: Improving Clinical Skills and Access Means Better Patient Care
by Patricia A Doorley and Charles G Durbin Jr—Charlottesville, Virginia **1015**

REVIEWS

Foreword: Thoracic Imaging in the Intensive Care Unit
by Eric J Stern—Seattle, Washington, and Robert D Tarver—Indianapolis, Indiana **1017**

Pitfalls in Portable Chest Radiology
by Heber MacMahon—Chicago, Illinois **1018**

Chest Imaging in the Cardiac Intensive Care Unit
by Ella A Kazerooni and Philip N Cascade—Ann Arbor, Michigan **1033**

Chest Imaging in the Trauma Intensive Care Unit
by Jannette Collins—Madison, Wisconsin **1044**

Chest Imaging in the Neurosciences Intensive Care Unit
by Daniel A Henry—Richmond, Virginia **1064**

Chest Imaging in the Surgical Intensive Care Unit
by Jeffrey S Klein—Burlington, Vermont **1078**

Chest Imaging in the Neonatal and Pediatric Intensive Care Units
by George W Gross—Wilmington, Delaware **1095**

Uses of Thoracic Computed Tomography in the Intensive Care Unit
by Wallace T Miller—Philadelphia, Pennsylvania **1127**

BOOKS, FILMS, TAPES, & SOFTWARE

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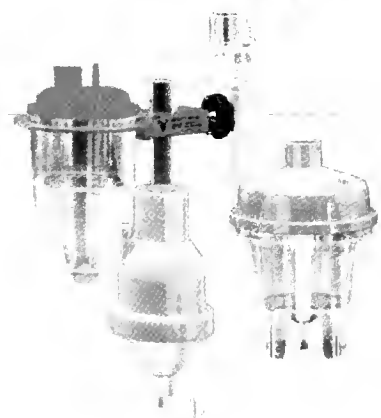

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CONTENTS

CONTINUED...

ALSO IN THIS ISSUE

1143 AARC Membership Application

1000 Abstracts from Other Journals

1152 Advertisers Index & Help Lines

1152 Author Index

1149 Calendar of Events

1145 Manuscript Preparation Guide

1141 Med Watch

1140 New Products & Services

1150 Notices

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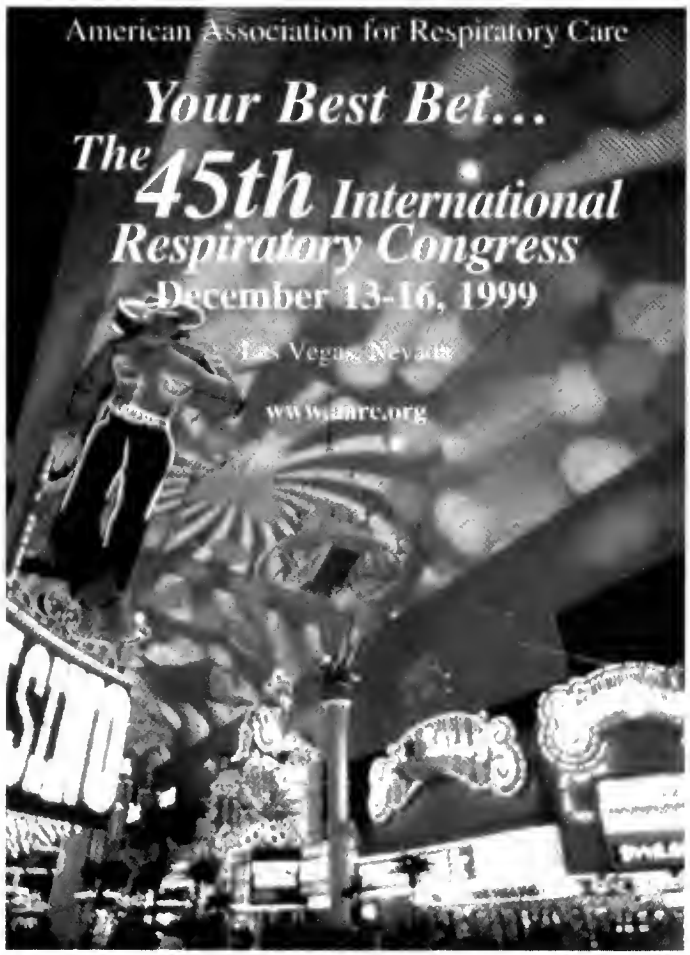

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Changes in Risk of Hospital Readmission Among Asthmatic Children in Denmark, 1978–93—Bisgaard H, Moller H. BMJ 1999 Jul 24;319(7204):229–230.

Difficult/Therapy-Resistant Asthma: The Need for an Integrated Approach to Define Clinical Phenotypes, Evaluate Risk Factors, Understand Pathophysiology and Find Novel Therapies. ERS Task Force on Difficult/Therapy-Resistant Asthma. European Respiratory Society—Chung KF, Godard P, Adelroth E, Ayres J, Barnes N, Barnes P, et al. Eur Respir J 1999 May;13(5):1198–1208.

Diurnal Variability: Time to Change Asthma Guidelines?—Reddel H, Jenkins C, Woolcock A. BMJ 1999 Jul 3;319(7201):45–47.

IV Immunoglobulin for Asthma?—Weinberger M. Chest 1999 Jul;116(1):267–268.

Self-Management Education for Adults with Asthma Improves Health Outcomes—Gibson PG, Coughlan J, Abramson M. West J Med 1999 May;170(5):266.

Third International Consensus Statement on the Management of Childhood Asthma—Konig P. Pediatr Pulmonol 1999 Jul;28(1):72–73.

What are Leukotrienes and How Do They Work in Asthma?—Berger A. BMJ 1999 Jul 10;319(7202):90.

The Burden of Asthma: Weighing the Community Risk Against Individual Risk—Von Schlegell A, Grant EN, Weiss KB. Thorax 1999 Jun;54(6):471–472.

Advising Parents of Asthmatic Children on Passive Smoking: Randomised Controlled Trial—Irvine L, Crombie IK, Clark RA, Slane PW, Feyerabend C, Goodman KE, Cater JI. BMJ 1999 May 29;318(7196):1456-1459.

Objective: To investigate whether parents of asthmatic children would stop smoking or alter their smoking habits to protect their children from environmental tobacco smoke. Design: Randomised controlled trial. Setting: Tayside and Fife, Scotland. Participants: 501 families with an asthmatic child aged 2-12 years living with a parent who smoked. Intervention: Parents were told about the impact of passive smoking on asthma and were advised to stop smoking or change their smoking habits to protect their child's health. Main outcome measures: Salivary cotinine concentrations in children, and changes in reported smoking habits of the parents 1 year after the intervention. Results: At the second visit, about 1 year after the baseline visit, a small decrease in salivary cotinine concentrations was found in both groups of children: the mean decrease in the intervention group (0.70 ng/mL) was slightly smaller than that of the control group (0.88 ng/mL), but the net difference of 0.19 ng/mL had a wide 95% confidence interval (-0.86 to 0.48). Overall, 98% of parents in both groups still smoked at follow up. However, there was a non-significant tendency for parents in the intervention group to report smoking more at follow up and to having a reduced desire to stop smoking. Conclusions: A brief intervention to advise parents of asthmatic children about the risks from passive smoking was ineffective in reducing their children's exposure to environmental tobacco smoke. The intervention may have made some parents less inclined to stop smoking. If a clinician believes that a child's health is being affected by parental smoking, the parent's smoking needs to be addressed as a separate issue from the child's health.

Inhaled Budesonide in Addition to Oral Corticosteroids to Prevent Asthma Relapse Following Discharge from the Emergency Department: A Randomized Controlled Trial—Rowe BH, Bota GW, Fabris L, Therrien SA, Milner RA, Jacono J. JAMA 1999 Jun 9;281(22):2119-2126.

CONTEXT: Relapses of acute asthma following emergency department (ED) discharge can be reduced with systemic corticosteroid treatment. However, whether inhaled corticosteroids (ICSs) provide additional benefit is not known. Objective To determine whether the addition of ICSs to oral corticosteroid treatment would reduce relapses in patients with acute asthma discharged from the ED. DESIGN AND SETTING: Placebo-controlled, double-blind, randomized clinical trial conducted in a community teaching hospital ED in Canada between November 1995 and September 1997, with a 21-day follow-up. PARTICIPANTS: A total of 1006 consecutive patients aged 16 to 60 years presented to the ED with acute asthma; after excluding those using oral and/or inhaled corticosteroids as well as those meeting other exclusion criteria, 188 were included in the study. INTER-

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VENTIONS: Patients were discharged with a nontapering course of oral prednisone (50 mg/d) for 7 days. In a double-blind fashion, patients were randomly assigned to 1600 microg/d of inhaled budesonide (n = 94) or identical placebo (n = 94) for 21 days. MAIN OUTCOME MEASURES: Incidence of relapse, defined as an unscheduled visit for worsening asthma symptoms, in budesonide vs placebo groups. Secondary outcomes included response to the Asthma Quality of Life Questionnaire, B2-agonist use, symptom score, global asthma improvement assessment, and pulmonary function. RESULTS: Five patients in the budesonide group and 3 in the placebo group either dropped out or were lost to follow-up but were included in primary analyses. After 21 days, 12 (12.8%) of 94 patients in the budesonide group experienced a relapse compared with 23 (24.5%) of 94 in the placebo group, a 48% relapse reduction (p=0.049). Asthma Quality of Life Questionnaire scores were higher (better quality) in the budesonide group (p=0.001), as well as for all domain scores (p=0.001 to 0.01). Fewer B2-agonist activations were used at the end of the trial by patients receiving budesonide (2.4/d vs 4.2/d; p=0.01). Symptom scores (p=0.001 to 0.004) and self-assessed asthma improvement scores (based on a 7-point Likert scale) (6.2 vs 5.2; p<0.001) were higher (indicating fewer symptoms) for budesonide vs placebo. There were no differences in pulmonary function between the groups (peak expiratory flow rate: budesonide, 437 vs placebo, 453 L/min; p= 0.39) at 21 days. Using this approach, as few as 9 patients would require budesonide to prevent 1 relapse. CONCLUSIONS: Patients discharged from the ED following treatment for acute asthma benefit from added treatment with high-dose inhaled budesonide for 21 days compared with oral corticosteroids alone.

Myopathy Following Mechanical Ventilation for Acute Severe Asthma: The Role of Muscle Relaxants and Corticosteroids—Behbehani NA, Al-Mane F, D'yachkova Y, Pare P, FitzGerald JM. Chest 1999 Jun;115(6):1627-1631.

BACKGROUND: Acute myopathy following mechanical ventilation for near-fatal asthma (NFA) has been described recently, and some researchers have suggested that this complication is related to the use of neuromuscular blocking agents (NMBAs) and corticosteroids (CSs). OBJECTIVES: To determine the incidence of acute myopathy in a group of patients and to examine the most important predictors of its development. DESIGN AND METHODS: A retrospective cohort study over a 10-year period (1985 to 1995) of all asthma patients who received mechanical ventilation at two centers in Vancouver (designated center 1 and center 2). RESULTS: In center 1, there were 58 patients who had 64 episodes of NFA, and in center 2, there were 28 patients who had 30 episodes. NMBAs were used in 30 of 86 admissions for acute severe asthma (35%). The mean (± SD) duration of muscle paralysis was 3.1±2.3 days. A total of 9 patients (10.4%) developed significant myopathy. The incidence of myopathy was 9 of 30 (30%) among patients who received NMBAs. In a multiple logistic regression model, the development of myopathy was only significantly associated with the duration of muscle relaxation. The odds ratio for the development of myopathy increased by 2.1 (95% confidence interval, 1.4 to 3.2) with each additional day of muscle relaxation. The dose and the type of the CS were not significantly associated with the myopathy in the multiple logistic regression analysis. CONCLUSION: Our study showed that there is a high incidence of acute myopathy when NMBAs are used for NFA. The incidence of myopathy increases with each additional day of muscle relaxation.

Asthma Symptoms: Influence of Personality Versus Clinical Status—Put C, Demedts M, Van Den Bergh O, Demyttenaere K, Verleden G. Eur Respir J 1999 Apr;13(4):751-756.

The hypothesis that symptom-reporting in asthmatics does not necessarily correspond with clinical status, but is related to negative affectivity was investigated. One hundred and sixteen asthmatic patients filled out the Asthma Symptom Checklist (ASC), the Negative Emotionality Scale (NEM), and the McMaster-Asthma Quality-of-Life Questionnaire (AQLQ). The patients were grouped as either hospitalized, outpatient previously hospitalized or outpatient not previously hospitalized for asthma. Lung function data and Asthma Severity Scores (ASS) were also collected. The hospitalized group was retested after 3 months. The hospitalized group had lower AQLQ scores, higher ASS scores and worse lung function than both outpatient groups. However, the hospitalized group and the outpatients previously hospitalized group had higher ASC scores and NEM scores than the outpatients not previously hospitalized group. After discharge, when the hospitalized group had become clinically stable, their ASC and NEM scores remained comparable to those of the outpatient previously hospitalized group. Symptom-reporting in asthmatics is not necessarily in accordance with clinical status, but may be directly or indirectly mediated by personality, such as negative affectivity.

Evaluation of a Randomised Controlled Trial of Adult Asthma Education in a Hospital Setting—Abdulwadud O, Abramson M, Forbes A,

James A, Walters EH. Thorax 1999 Jun;54(6): 493-500.

BACKGROUND: Although patient education is a key step in the Australian Asthma Management Plan, its impact has not been assessed in a hospital outpatient asthma clinic. METHODS: A controlled trial was undertaken in 125 adults with asthma recruited from the Alfred Hospital Asthma and Allergy Clinic and randomly allocated to an intervention (n = 64) or control (n = 61) group. Subjects in the intervention group attended three education sessions, each of 90 minutes duration, spread over three successive weeks. Asthma knowledge, quality of life, self-management skills, and attitudes and beliefs about asthma were assessed by questionnaires at baseline and after six months. The intervention group was also assessed immediately after the three education sessions. The control group was evaluated after six months of usual care. RESULTS: Asthma knowledge improved significantly in the intervention group after three education sessions ($p = 0.0001$) and this was retained six months later ($p = 0.03$). The impact of asthma on quality of life decreased significantly immediately after intervention ($p = 0.03$) but this was not maintained six months later ($p = 0.35$). On the other hand, the intervention had little impact on self-management skills or attitudes and beliefs about asthma. However, the control group had also improved their knowledge, quality of life and self-management skills after six months of usual care. The difference in mean change in knowledge score at six months between the intervention and control groups was not significant ($p = 0.51$). CONCLUSIONS: In contrast to some other studies, a limited asthma education programme in a hospital outpatient setting had a positive impact on patients' knowledge of asthma, but not on their quality of life, self-management skills, or attitudes and beliefs about asthma.

Physiologic Response of Ventilator-Dependent Patients with Chronic Obstructive Pulmonary Disease to Proportional Assist Ventilation and Continuous Positive Airway Pressure—Appendini L, Purro A, Gudjonsdottir M, Baderna P, Patessio A, Zanaboni S, Donner CF, Rossi A. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1510-1517.

To investigate the physiologic effects of proportional assist ventilation (PAV) in difficult-to-wean, mechanically ventilated patients with advanced COPD, we measured in eight ICU patients the breathing pattern, neuromuscular drive ($P_{0.1}$), lung mechanics, and inspiratory muscle effort (PTPdi and PTPpl) during both spontaneous breathing (SB) and ventilatory support with PAV, CPAP, and CPAP + PAV (in random sequence). PAV (volume assist [VA] and flow assist [FA]) was set as follows: dynamic lung elastance and inspiratory pulmonary resistance were measured during SB; then VA and FA were set to counterbalance the elastic and resistive loads exceeding the normal values, respectively, the inspiratory muscles bearing a normal elastic and resistive workload. CPAP was set close to dynamic intrinsic PEEP (8.3 ± 3.4 cm H_2O). We found significant reductions in $P_{0.1}$ and PTPdi during both CPAP (-45 and -37%, respectively) and PAV (-50 and -48%, respectively). However, only the combination of PAV and CPAP brought $P_{0.1}$ (1.69 ± 0.97 cm H_2O) and PTPdi (100 ± 68 cm $H_2O \cdot s$) within normal values, and ameliorated the breathing pattern compared with SB (tidal volume: 0.69 ± 0.33 versus 0.33 ± 0.14 L; breathing frequency, 14.6 ± 4.6 versus 21.0 ± 6.5 breaths/min, respectively), without generating ineffective inspiratory efforts. We conclude that in difficult-to-wean COPD patients, (1) PAV improves ventilation and reduces both P0.1 and inspiratory muscle effort; (2) the combination of PAV and CPAP can unload the inspiratory muscles to values close to those found in normal subjects.

Outcomes After Long-Term Acute Care: An Analysis of 133 Mechanically Ventilated Patients—Carson SS, Bach PB, Brzozowski L, Leff A. Am J Respir Crit Care Med 1999 May; 159(5 Pt 1):1568-1573.

Long-term acute-care (LTAC) hospitals are facilities exempt from the Medicare prospective payment system and which provide care to patients suffering from prolonged critical illness. From August 1, 1995 to July 31, 1996, we studied the outcomes of 133 mechanically ventilated patients who were consecutively admitted to a large urban LTAC hospital from intensive care units (ICUs) of acute-care hospitals. Survival and functional status within 1 yr after the index admission were measured, and specific patient variables were used to develop a predictive model for survival at 1 yr. Of the 133 patients studied, 66 (50%) died prior to discharge. Of discharged patients, 70% had been successfully liberated from mechanical ventilation. One year after LTAC hospital admission, 103 (77%) of the patients had expired, typically after spending the majority of their days in acute care or long-term care facilities. Eleven 1-yr survivors (8%) were fully functional, whereas the remainder had significantly reduced functional status. Patients older than 74 yr, and patients older than 64 yr and not functionally independent before admission, had a 95% (confidence interval [CI]: 84% to 99%) 1-yr mortality; patients without these characteristics had a 56% (CI: 41% to 71%) 1-yr mortality ($p < 0.001$). We demonstrate characteristics predicting the poorest prognoses for patients requiring prolonged mechanical ventilation. These characteristics may be identifiable before transfer to an LTAC hospital.

A Scanographic Assessment of Pulmonary Morphology in Acute Lung Injury: Significance of the Lower Inflection Point Detected on the Lung Pressure-Volume Curve—Vieira SR, Puybasset L, Lu Q, Richecoeur J, Cluzel P, Coriat P, Rouby JJ. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1612-1623.

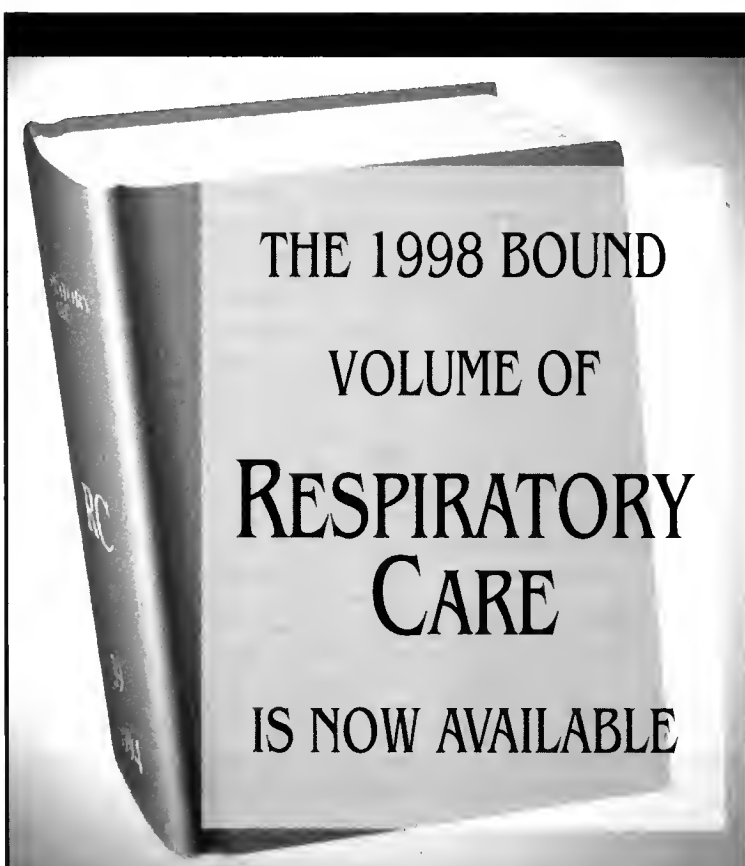
The goal of this study was to assess lung morphology in patients with acute lung injury according to the presence or the absence of a lower inflection point (LIP) on the lung pressure-volume (P-V) curve and to compare the effects of positive end-expiratory pressure (PEEP). Eight patients with and six without an LIP underwent a spiral thoracic CT scan performed at zero end-expiratory pressure (ZEEP) and at two levels of PEEP: PEEP1 = LIP + 2 cm H_2O and PEEP2 = LIP + 7 cm H_2O, or PEEP1 = 10 cm H_2O and PEEP2 = 15 cm H_2O in the absence of an LIP. The volumes of air and tissue within the lungs were measured from the gas-tissue ratio and the volumes of overdistended and normally, poorly, and nonaerated lung areas were determined by the analysis of the frequency histogram distribution. In the ZEEP condition, although total lung volume, volume of gas, and volume of tissue were similar in both groups, the percentage of normally aerated lung was lower ($24 \pm 22\%$ versus $55 \pm 12\%$, $p < 0.05$) and the percentage of poorly aerated lung was greater ($40 \pm 12\%$ versus $23 \pm 8\%$, $p < 0.05$) in patients with an LIP than in patients without an LIP. Lung density histograms of patients with an LIP showed a unimodal distribution with a peak at 7 Hounsfield units (HU). Lung density histograms of patients without an LIP had a bimodal distribution, with a first peak at -727 HU and a second peak at 27 HU. Total respiratory system and lung compliances were lower in patients with an LIP whereas all other cardiorespiratory parameters were similar in the two groups. In both groups, PEEP induced an alveolar recruitment that was associated with lung overdistension only in patients without an LIP. The amount of lung overdistension was related to the volume of lung parenchyma, characterized by a CT number less than -800 HU before PEEP implementation ($y = 0.52x + 4$, $r = 0.87$, and $p < 0.0001$). This study shows that the presence or the absence of an LIP on the lung P-V curve is associated with differences in lung morphology. In patients without an LIP on the lung P-V curve, normally aerated lung areas coexist with nonaerated lung areas and increasing levels of PEEP result in lung overdistension rather than in additional alveolar recruitment. In patients with an LIP, air and tissue are more homogeneously distributed within the lungs and increasing levels of PEEP result in additional alveolar recruitment without lung overdistention.

The FEF_{25-75}/FVC Ratio Is Associated with Methacholine Airway Responsiveness: The Normative Aging Study—Litonjua AA, Sparrow D, Weiss ST. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1574-1579.

Airway responsiveness is known to be partly explained by geometric and anatomic factors. This cross-sectional investigation was undertaken to determine whether FEF_{25-75}/FVC as a surrogate measure of airway size relative to lung size is associated with airway responsiveness to methacholine. Posteroanterior chest radiographs and spirometry were performed on a group of 929 middle aged and older men from an ongoing longitudinal study, the Normative Aging Study, who returned for their regularly scheduled examination between 1984 and 1989. Subjects had a mean age of 60.5 ± 7.7 yr. FEV_1, FEF_{25-75}, and FVC were taken from spirometric results and FEF_{25-75}/FVC ratios were obtained. Main bronchus (MB) and tracheal (TR) diameters and lung area (LA) were obtained from chest radiographs, and ratios of MB/LA and TR/LA were calculated for each subject and compared with FEF_{25-75}/ FVC as measures of airway size relative to lung size. In a multiple linear regression model adjusting for age, height, initial FEV_1, smoking, eosinophil count, and IgE level, FEF_{25-75}/FVC was significantly related to the degree of methacholine airway responsiveness as measured by Log10 dose response slope ($B = -0.37$, $p < 0.001$). Controlling for the same variables, both MB/LA ($B = -149.07$, $p < 0.001$) and TR/LA ($B = -125.87$, $p < 0.001$) were significant predictors of the degree of bronchial responsiveness in separate regression models; however, their effects were greatly attenuated when FEF_{25-75}/ FVC was present in the same model. Similar results were obtained after excluding subjects with FEV_1/ FVC ≤ 0.70 and subjects who had any smoking history. We conclude that FEF_{25-75}/FVC as a surrogate measure of airway size relative to lung size is significantly associated with airway responsiveness.

Prospective Evaluation of an Algorithm for the Functional Assessment of Lung Resection Candidates—Wyser C, Stulz P, Soler M, Tamm M, Muller-Brand J, Habicht J, Perruchoud AP, Bolliger CT. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1450-1456.

Patients with impaired pulmonary function are at increased risk for the development of postoperative complications. Recently exercise testing and predicted postoperative (ppo) function have gained increasing importance in the evaluation of lung resection candidates. We prospectively evaluated an algorithm for the preoperative functional evaluation that was developed at our institution. This algorithm incorporated the cardiac history including an electrocardiogram (ECG), and the three parameters FEV_1, diffusing capacity of the lungs for carbon monoxide (DLCO), and maximal oxygen uptake (V_{O_2}max), as well as their respective ppo values (FEV_1-ppo, DLCO-ppo, and V_{O_2}max-ppo) calculated based on radionuclide perfusion scans. A consecutive group of 137 patients (mean age 62 yr; range 23 to 81; 102 males, 35 females) with clinically resectable lesions underwent assessment according to our algorithm. Five patients were deemed functionally inoperable. 132 passed the algorithm and underwent pulmonary resections with standard thoracotomy: 9 segmental or wedge resections, 85 lobectomies (inclusive 3 bilobectomies), and 38 pneumonectomies. All patients were extubated within 24 h. The mean stay in the ICU was 1.4 (± 1.8) d, and the mean hospital stay was 14.6 (± 5) d. Postoperative complications (within 30 d) occurred in 15 patients (11%), of whom two died (overall mortality rate 1.5%). In comparison to our previous series this meant a 50% reduction in complications whereas the percentage of inoperable patients remained unchanged (4% now, 5% before). We conclude that adherence to our algorithm resulted in a



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very low complication rate (morbidity and mortality), and excluded more rigorous patient selection as a bias for the improved results.

Equivalent Bronchodilation with Salbutamol Given via pMDI or Turbuhaler—Mellen A, Arvidsson P, Palmqvist M, Lotvall J. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1663-1665.

Several breath-activated multidose powder devices for inhaled anti-asthma drugs are now available. Some of these inhalers have been argued to give higher drug deposition in the airways than conventional pressurized metered dose inhalers (pMDI). The aim of the present study was to compare the efficacy and safety of salbutamol given via pMDI or Turbuhaler (both 100 microgram per inhalation). Adult asthmatic patients of either sex (n = 22) and with reversible airflow obstruction were included in a randomized, placebo-controlled study. On the study days, salbutamol was given with increasing doses (200 to 3,200 microgram cumulative) or placebo, via pMDI or Turbuhaler. A dose-related increase in FEV_1 was observed after administration of salbutamol given via either device, versus placebo. The improvement in FEV_1 was similar whether salbutamol was given via pMDI or Turbuhaler, at microgram equivalent doses. After a total cumulative dose of 3,200 microgram, mean FEV_1 for Turbuhaler was 2.98 (change from baseline of 23.1%), for pMDI 2.93 (change from baseline of 23.6%), and for placebo 2.36 (change from baseline of 0.42%). Changes in potassium, glucose, and heart rate did not show any significant differences between pMDI and Turbuhaler. We conclude that the efficacy of salbutamol is comparable when the drug is given via the Turbuhaler or pMDI.

Relationship Between Resting Hypercapnia and Physiologic Parameters Before and After Lung Volume Reduction Surgery in Severe Chronic Obstructive Pulmonary Disease—Shade D Jr, Cordova F, Lando Y, Travaline JM, Furukawa S, Kuzma AM, Criner GJ. Am J Respir Crit Care Med 1999 May; 159(5 Pt 1):1405-1411.

Patients with severe chronic obstructive pulmonary disease (COPD) have varying degrees of hypercapnia. Recent studies have demonstrated inconsistent effects of lung volume reduction surgery (LVRS) on P_{aCO_2}; however, most series have excluded patients with moderate to severe hypercapnia. In addition, no study has examined the mechanisms responsible for the reduction in P_{aCO_2} post-LVRS. We obtained spirometry, body plethysmography, diffusion capacity, respiratory muscle strength, 6-min walk test, and incremental symptom-limited maximal exercise data in 33 consecutive patients pre- and 3 to 6 mo post-LVRS, and explored the relationship between changes in P_{aCO_2} and changes in the measured physiologic variables. All patients underwent bilateral LVRS via median sternotomy and stapling resection by the same cardiothoracic surgeon. Patients were 57 ± 8 yr of age with severe COPD, hyperinflation, and air trapping (FEV_1, 0.73 ± 0.2 L; TLC, 7.3 ± 1.6 L; residual volume [RV], 4.8 ± 1.4 L), and moderate resting hypercapnia (P_{aCO_2}, 44 ± 7 mm Hg; range, 32 to 56 mm Hg). Post-LVRS, P_{aCO_2} decreased by 4% (P_{aCO_2} pre 44 ± 7 mm Hg, P_{aCO_2} post 42 ± 5 mm Hg; p = 0.003). Patients with higher baseline values of P_{aCO_2} had the greatest reduction in P_{aCO_2} post-LVRS ($r = -0.61$, $p < 0.001$). Significant correlations existed between reduction in P_{aCO_2} and changes in FEV_1 ($r = -0.56$; $p = 0.0007$), maximal inspiratory pressure (PImax) ($r = -0.46$; $p = 0.009$), diffusing capacity of the lungs for carbon monoxide (DLCO) ($r = -0.47$; $p = 0.008$), and RV/TLC ($r = 0.41$; $p = 0.02$). Correlation existed also between reduction in P_{aCO_2} and breathing pattern at maximal exercise: maximal minute ventilation ($\dot{V}_E$max) ($r = -0.47$; $p = 0.009$), and tidal volume (V_T) ($r = -0.40$; $p = 0.02$). The changes in P_{aCO_2} post-LVRS showed marked intersubject variability. We conclude that LVRS, by reducing hyperinflation, air trapping, and improving respiratory muscle function, enables the lung and chest wall to act more effectively as a pump, thereby increasing alveolar ventilation and reducing baseline resting P_{aCO_2}. In addition, patients with higher baseline levels of P_{aCO_2} demonstrate the greatest reduction in P_{aCO_2} post-LVRS, and should not be excluded from receiving LVRS.

Target Dyspnea Ratings Predict Expected Oxygen Consumption As Well As Target Heart Rate Values—Mejia R, Ward J, Lentine T, Mahler DA. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1485-1489.

A target heart rate (THR) is the traditional method to prescribe and monitor exercise training intensity in healthy individuals. However, patients with chronic obstructive pulmonary disease (COPD) are limited by ventilatory impairment and dyspnea rather than cardiovascular factors. An alternative approach is to use dyspnea ratings as a target for exercise training in patients with respiratory disease just as ratings of perceived exertion have been used in healthy individuals. The study was a randomized, parallel group trial comparing the ability of patients with COPD to accurately and reliably produce an exercise intensity using a target dyspnea rating (TDR) versus a THR. At Visit 1 patients performed an incremental exercise test on the cycle ergometer, and target values were calculated at approximately 75% of maximal oxygen consumption ($\dot{V}_{O_2}$). At Visits 2 (3 to 5 d later) and 3 (2 wk later) each patient was instructed to produce a TDR or a THR for 10 min of submaximal exertion. Anthropometric characteristics, lung function, and exercise performance were similar for the 22 patients in each group at Visit 1. For the TDR group the dyspnea target was 2.5 ± 1.5 at an expected $\dot{V}_{O_2}$ of 0.88 ± 0.28 L/min; for the THR group the heart rate (HR) target was 114 ± 15 beats/min at an expected $\dot{V}_{O_2}$ of 0.76 ± 0.29 L/min ($p = 0.18$ for $\dot{V}_{O_2}$ between groups). Compared with the expected $\dot{V}_{O_2}$ from Visit 1, the individual percent differences in $\dot{V}_{O_2}$ at Visit 2 were -3.9 ± 18.1% (TDR) and -0.5 ± 23.2% (THR) ($p = 0.58$); at Visit 3 the individual percent differences in $\dot{V}_{O_2}$ were -2.3 ± 17.0% (TDR) and 2.6 ± 30.6% (THR) ($p = 0.52$). The number of patients < 10% and ≥ 10% of the expected $\dot{V}_{O_2}$ were similar for the two groups at Visits 2 ($p = 0.38$) and 3 ($p = 0.27$). There were no significant differences for $\dot{V}_{O_2}$ values (absolute or individual percent) at Visits 2 and 3 for each group and between the groups ($p = 0.79$). In conclusion, patients with symptomatic COPD demonstrated a comparable ability to use dyspnea ratings and HR as a target to accurately and reliably produce an expected exercise intensity (approximately 75% of $\dot{V}_{O_2}$max) for 10 min of submaximal exertion.

Effects of Inhaled Carbon Dioxide and Oxygen on Cheyne-Stokes Respiration in Patients with Heart Failure—Lorenzi-Filho G, Rankin F, Bies I, Bradley DT. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1490-1498.

We hypothesized that in patients with congestive heart failure (CHF), reductions in P_{aCO_2} sensed at the peripheral chemoreceptors trigger central apneas during Cheyne-Stokes respiration (CSR-CSA), and that raising P_{aCO_2} by inhalation of a CO_2 would eliminate these events. The effects of CO_2 inhalation on the frequency of apneas and hypopneas during stage 2 (S2) sleep were studied in 10 CHF patients with CSR-CSA. The time from the breath with the minimal end tidal fraction of CO_2 (F_{ETCO_2}) during hyperpnea until the onset of apnea correlated strongly with the lung to ear circulation time (LECT) ($r^2 = 0.90$, $p < 0.0001$), a measure of lung to carotid body circulatory delay. Among the six patients who also inhaled O_2, CO_2 inhalation increased transcutaneous PCO_2 ($PtcCO_2$) (36.4 ± 4.6 mm Hg versus 38 ± 4.4 mm Hg, $p < 0.002$), abolished central apneas and hypopneas (43.0 ± 8.4 per hour on air versus 1.6 ± 2.6 per hour on CO_2, $p < 0.0001$), and increased S_{aO_2}. In contrast, O_2 inhalation causing a similar rise in S_{aO_2} had no significant impact on either $PtcCO_2$ or the frequency of central events. We conclude that central apneas in patients with CHF are triggered by a low P_{aCO_2} most likely sensed at the peripheral chemoreceptors, and that inhalation of CO_2 reverses central apneas by raising P_{aCO_2}.

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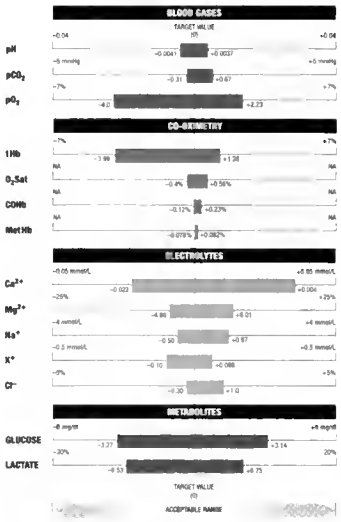

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Lung Disease—Sajkov D, Wang T, Saunders NA, Bune AJ, Neill AM, Douglas Mcevoy R. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1518-1526.

It is controversial whether obstructive sleep apnea (OSA) causes pulmonary hypertension (PH) in the absence of hypoxemic lung disease. To investigate this further we measured awake pulmonary hemodynamics, pulmonary gas exchange, and small airways function in 32 patients with OSA (apnea- hypopnea index, mean ± SE, 46.2 ± 3. 9/h) who had normal screening lung function. Pulmonary artery pressure (Ppa) and cardiac output were measured by Doppler echocardiography at three levels of inspired oxygen (F_{IO_2} 0.50, 0.21, and 0.11) and during incremental increases in pulmonary blood flow (10, 20, and 30 microgram/kg/min dobutamine infusions) while breathing 50% oxygen. Eleven patients had PH (mean Ppa ≥ 20 mm Hg, Group I). They did not differ from patients without PH (Group II) in lung function, severity of sleep-disordered breathing, age, or body mass. Compared with Group II, Group I patients had increased small airways closure during tidal breathing (FRC-closing capacity: Group I, -0.16 ± 0.11; Group II, 0.27 ± 0.09 L; $p < 0.05$), more ventilation-perfusion inequality ($AaPO_2$: 23.8 ± 2.8; 19.8 ± 1.4 mm Hg; $p = 0.08$), a greater pulmonary artery pressor response to hypoxia (ΔPpa F_{IO_2}, 0.50 to 0.11: 16.4 ± 1.93; 6.4 ± 0.77 mm Hg; $p < 0.05$) and a marked rise in Ppa during increased pulmonary blood flow. We conclude that PH may develop in some patients with OSA without lung disease and that it is associated with small airways closure during tidal breathing and heightened pulmonary pressor responses to hypoxia and during increased pulmonary blood flow. Such changes are consistent with remodeling of the pulmonary vascular bed in affected patients with OSA, seemingly unrelated to severity of sleep-disordered breathing.

Cough Reflex Sensitivity in Subjects with Cervical Spinal Cord Injury—Dicpinigaitis PV, Grimm DR, Lesser M. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1660-1662.

An effective cough requires an intact cough reflex as well as adequate respiratory muscle function to generate elevated intrathoracic pressures. Since the major muscles of expiration are innervated by the first thoracic segment and below, transection of the cervical spinal cord results in severely compromised expiratory function and cough. To investigate the effects of cervical spinal cord injury (C-SCI) on cough reflex sensitivity, we measured responsiveness to inhaled capsaicin in 12 male subjects with chronic C-SCI and compared findings to those from a control group of 50 able-bodied men. The concentrations (microM) of capsaicin inducing two or more (C2) and five or more coughs (C5) did not significantly differ between the two groups. Mean (± SEM) values for log C2 in subjects with C-SCI and control subjects were 0.65 ± 0.15 and 0.87 ± 0.07, respectively ($p = 0.15$). Mean values for log C5 in subjects with C-SCI and control subjects were 1.43 ± 0.23 and 1.41 ± 0.08, respectively ($p = 0.94$). We conclude that cough reflex sensitivity is preserved after C-SCI, and that ineffective cough in this population results primarily from the loss of innervation of respiratory muscles.

Risk Factors for Sleep-Disordered Breathing in Children: Associations with Obesity, Race, and Respiratory Problems—Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1527-1532.

This study examined risk factors for sleep-disordered breathing (SDB) in children and adolescents; specifically, quantifying risk associated with obesity, race, and upper and lower respiratory problems. Subjects were participants in a genetic-epidemiologic study of SDB and included 399 children and adolescents 2 to 18 yr of age, recruited as members of families with a member (a proband) with known sleep apnea (31 index families) or as members of neighborhood control families (30 families). SDB was assessed with home overnight multichannel monitoring and SDB was defined based on an apneahypopnea index ≥ 10 (moderately affected) or < 5 (unaffected). SDB of moderate level was significantly associated with obesity (odds ratio, 4.59; 95% confidence interval [CI], 1.58 to 13.33) and African-American race (odds ratio, 3.49; 95% CI, 1.56 to 8.32) but not with sex or age. After adjusting for obesity, proband sampling, race and familial clustering, sinus problems and persistent wheeze each independently (of the other) predicted SDB. These data suggest the importance of upper and lower respiratory problems and obesity as risk factors for SDB in children and adolescents. Increased risk in African Americans appears to be independent of the effects of obesity or respiratory problems.

Dysfunctional Mechanical Coupling of Upper Airway Tissues in Sleep Apnea Syndrome—Series F, Cote C, St Pierre S. Am J Respir Crit Care Med 1999 May;159(5 Pt 1): 1551-1555.

The mechanical effect of musculus uvulae (MU) contraction on in vitro uvular shortening and/or displacement was measured in 15 patients with a sleep apnea hypopnea syndrome (SAHS) and in 8 snorers. Using freshly resected uvular tissues, passive and active uvular tissue-specific elastance and shortening were determined during stimulation of MU. No difference was found in maximum tetanic tension measured on uvular tissue between the two groups (47.2 ± 14.8 g in SAHS and 39.1 ± 16.5 g in snorers). Passive uvular-specific elastance was significantly less in snorers (0.36 ± 0.27 g/% Lo) compared with patients with SAHS (0.84 ± 0.39 g/% Lo) ($p = 0.006$). There was a negative correlation between uvular shortening and passive uvular specific elastance ($r = 0.69$, $p = 0. 05$). Maximal tetanic tension developed by isolated MU was higher in SAHS than in snorers (45.8 ± 23.1 and 30.0 ± 8.3 g, respectively, $p = 0.04$). A strong positive relationship was found between the apnea index and specific uvular elastance ($r = 0.55$, $p = 0.007$). We conclude that there is a significant difference between the uvular tissue elastance of SAHS and snorers, and that this difference influences the mechanical efficiency of MU contraction.

The Unreliability of Individual Physician 'Report Cards' for Assessing the Costs and Quality of Care of a Chronic Disease—Hofer TP, Hayward RA, Greenfield S, Wagner EH, Kaplan SH, Manning WG. JAMA 1999 Jun 9;281(22): 2098-2105.

CONTEXT: Physician profiling is widely used by many health care systems, but little is known about the reliability of commonly used profiling systems. OBJECTIVES: To determine the reliability of a set of physician performance measures for diabetes care, one of the most common conditions in medical practice, and to examine whether physicians could substantially improve their profiles by preferential patient selection. DESIGN AND SETTING: Cohort study performed from 1990 to 1993 at 3 geographically and organizationally diverse sites, including a large staff-model health maintenance organization, an urban university teaching clinic, and a group of private-practice physicians in an urban area. PARTICIPANTS: A total of 3642 patients with type 2 diabetes cared for by 232 different physicians. MAIN OUTCOME MEASURES: Physician profiles for their patients' hospitalization and clinic visit rates, total laboratory resource utilization rate and level of glycemic control by average hemoglobin A1c level with and without detailed case-mix adjustment. RESULTS: For profiles based on hospitalization rates, visit rates, laboratory utilization rates, and glycemic control, 4% or less of the overall variance was attributable to differences in physician practice and the reliability of the median physician's case-mix-adjusted profile was never better than 0.40. At this low level of physician effect, a physician would need to have more than 100 patients with diabetes in a panel for profiles to have a reliability of 0.80 or better (while more than 90% of all primary care physicians at the health maintenance organization had fewer than 60 patients with diabetes). For profiles of glycemic control, high outlier physicians could dramatically improve their physician profile simply by pruning from their panel the 1 to 3 patients with the highest he-

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moglobin A1c levels during the prior year. This advantage from gaming could not be prevented by even detailed case-mix adjustment. CONCLUSIONS: Physician 'report cards' for diabetes, one of the highest-prevalence conditions in medical practice, were unable to detect reliably true practice differences within the 3 sites studied. Use of individual physician profiles may foster an environment in which physicians can most easily avoid being penalized by avoiding or deselecting patients with high prior cost, poor adherence, or response to treatments. *See the related editorial:* ***Can Physician Profiles Be Trusted?*** *Bindman AB. JAMA 1999 Jun 9;281(22):2142-2143.*

Nosocomial Infections in Medical Intensive Care Units in the United States. National Nosocomial Infections Surveillance System—Richards MJ, Edwards JR, Culver DH, Gaynes RP. Crit Care Med 1999 May;27(5):887-892.

OBJECTIVE: To describe the epidemiology of nosocomial infections in medical intensive care units (ICUs) in the United States. DESIGN: Analysis of ICU surveillance data collected through the National Nosocomial Infections Surveillance (NNIS) System between 1992 and 1997. SETTING: Medical ICUs in the United States. PATIENTS: A total of 181,993 patients. MEASUREMENTS AND MAIN RESULTS: Nosocomial infections were analyzed by infection site and pathogen distribution. Urinary tract infections were most frequent (31%), followed by pneumonia (27%) and primary bloodstream infections (19%). Eighty-seven percent of primary bloodstream infections were associated with central lines, 86% of nosocomial pneumonia was associated with mechanical ventilation, and 95% of urinary tract infections were associated with urinary catheters. Coagulase-negative staphylococci (36%) were the most common bloodstream infection isolates, followed by enterococci (16%) and *Staphylococcus aureus* (13%). Twelve percent of bloodstream isolates were fungi. The most frequent isolates from pneumonia were Gram-negative aerobic organisms (64%). *Pseudomonas aeruginosa* (21%) was the most frequently isolated of these. *S. aureus* (20%) was isolated with similar frequency. *Candida albicans* was the most common single pathogen isolated from urine and made up just over half of the fungal isolates. Fungal urinary infections were associated with asymptomatic funguria rather than symptomatic urinary tract infections ($p < .0001$). Certain pathogens were associated with device use: coagulase-negative staphylococci with central lines, *P. aeruginosa* and Acinetobacter species with ventilators, and fungal infections with urinary catheters. Patient nosocomial infection rates for the major sites correlated strongly with device use. Device exposure was controlled for by calculating device-associated infection rates for bloodstream infections, pneumonia, and urinary tract infections by dividing the number of device-associated infections by the number of days of device use. There was no association between these device-associated infection rates and number of hospital beds, number of ICU beds, or length of stay. There is a considerable variation within the distribution of each of these infection rates. CONCLUSIONS: The distribution of sites of infection in medical ICUs differed from that previously reported in NNIS ICU surveillance studies, largely as a result of anticipated low rates of surgical site infections. Primary bloodstream infections, pneumonia, and urinary tract infections associated with invasive devices made up the great majority of nosocomial infections. Coagulase-negative staphylococci were more frequently associated with primary bloodstream infections than reported from NNIS ICUs of all types in the 1980s, and enterococci were a more frequent isolate from bloodstream infections than *S. aureus*. Fungal urinary tract infections, often asymptomatic and associated with catheter use, were considerably more frequent than previously reported. Invasive device-associated infections were associated with specific pathogens. Although device-associated site-specific infection rates are currently our most useful rates for performing comparisons between ICUs, the considerable variation in these rates between ICUs indicates the need for further risk adjustment.

Do the Components of Heat and Moisture Exchanger Filters Affect Their Humidifying Efficacy and the Incidence of Nosocomial Pneumonia?—Thomachot L, Vialet R, Arnaud S, Barberon B, Michel-Nguyen A, Martin C. Crit Care Med 1999 May;27(5):923-928.

OBJECTIVES: To compare the efficiency of two heat and moisture exchange filters (HMEFs) of different compositions of the humidifying capacity and the rate of bronchial colonization and ventilator-associated pneumonia in patients in the intensive care unit (ICU). DESIGN: Prospective, randomized study. SETTING: ICU of a university hospital. PATIENTS: All patients who required mechanical ventilation for 24 hrs or more during the study period. INTERVENTIONS: At admission to the ICU, patients were randomly assigned to one of two groups. In one group, the patients were ventilated with Humid-Vent Filter Light HMEF. The condensation surface was made of paper impregnated with $CaCl_2$. The filter membrane was made of polypropylene. In the other group, the patients were ventilated with the Clear ThermAl HMEF (Intersurgical, France). The condensation surface was made of plastic foam impregnated with $AlCl_2$. The filter membrane was made of two polymer fibers (modacrylic and polypropylene). In both groups, HMEFs were changed daily. MEASUREMENTS AND MAIN RESULTS: Seventy-seven patients were ventilated for 19±7 days with the Humid-Vent Filter Light HMEF and 63 patients for 17±6 days with the Clear ThermAl HMEF. Patients ventilated with the Humid-Vent Filter Light underwent 8.7±3.7 tracheal aspirations and 1.2±2.0 instillations per day and those with the Clear ThermAl, 8.2±3.9 and 1.5±2.4 per day, respectively (NS). The abundance of tracheal secretions and the presence of blood and viscosity, as evaluated by semiquantitative scales, were similar in both groups. One episode of tracheal tube occlusion was observed with the Humid-Vent Filter Light HMEF and none with the other HMEF (NS). Tracheal colonization was observed at a rate of 91% with the Humid-Vent Filter Light and 97% with the Clear ThermAl (NS). The rate of ventilator-associated pneumonia was similar in both groups (35%). Bacteria responsible for tracheal colonization and pneumonia were similar in both groups. CONCLUSIONS: Despite differences in their components, the two HMEFs that were tested achieved similar performances in terms of humidification and heating of inspired gases. Only one episode of endotracheal tube occlusion was detected, and very few patients (three in each group) had to be switched to an active heated humidifier. No difference was observed either in the rate of tracheal colonization or of ventilator-associated pneumonia. These data show that the Humid-Vent Filter Light and the Clear ThermAl HMEFs are suited for use with ICU patients.

Prolonged Partial Liquid Ventilation in Spontaneously Breathing Awake Animals—Rich PB, Reickert CA, Mahler SA, Sawada S, Awad S, Kazerooni EA, Hirschl RB. Crit Care Med 1999 May;27(5):941-945.

OBJECTIVE: To date, studies of partial liquid ventilation (PLV) have examined its effects acutely in anesthetized and mechanically ventilated subjects. We set out to develop a model of prolonged PLV in awake, spontaneously breathing animals. DESIGN: Animal case series SETTING: Cardiopulmonary physiology laboratory. SUBJECTS: Fifteen New Zealand white rabbits (3.2±0.39 kg). INTERVENTIONS: Animals were anesthetized and instrumented with a novel technique allowing percutaneously assisted placement of an intratracheal catheter with a subcutaneously tunneled externalized free end. After anesthetic recovery, PLV was performed in spontaneously breathing unsedated animals. MEASUREMENTS AND MAIN RESULTS: Evaporative losses were determined using a single 10 mL/kg perflubron dose ($n = 5$); hourly radiographs were obtained until residual perflubron was minimal. For prolonged PLV ($n = 10$), a 10-mL/kg initial perflubron dose was followed every 4 hrs with 5-mL/kg supplements. Radiographs were obtained immediately before and after perflubron administration and were scored (0-5) by a radiologist blinded to dosing regimen and time interval. Data were analyzed with ANOVA and

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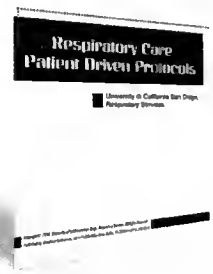

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Student's t-test with correction for multiple comparisons. Initial filling was nearly complete (score = 4.8±0.42); lungs were maintained approximately half-filled through 4 hrs (score = 2.53±0.71). By 6 hrs, the majority of perflubron had evaporated (score = 1.75±0.53). Over 24 hrs, radiographs documented continuous perflubron exposure (postffill = 4.53±0.64, prefill = 3.40±0.71, average = 3.97±0.43); scores were comparatively higher after filling (after = 4.53±0.64, before = 3.4±0.71, $p < 0.001$). Left and right lung volumes were equivalent (left = 4.06±0.47, right = 3.89±0.39, $p = 0.027$). All animals survived the 24 hrs of PLV. CONCLUSIONS: Percutaneously assisted intratracheal cannulation with catheter exteriorization permits prolonged PLV in spontaneously breathing, unsedated animals. Continuous perfluorocarbon exposure with this method is reproducible, consistent, and well tolerated for 24 hrs in uninjured animals.

Phase-Related Changes in Right Ventricular Cardiac Output Under Volume-Controlled Mechanical Ventilation with Positive End-Expiratory Pressure—Theres H, Binkau J, Laule M, Heinze R, Hundertmark J, Blobner M, et al. Crit Care Med 1999 May;27(5):953-958.

OBJECTIVE: To examine determinants of right ventricular function throughout the ventilatory cycle under volume-controlled mechanical ventilation with various positive end-expiratory pressure (PEEP) stages. DESIGN: Prospective observational animal pilot study. SETTING: Animal research laboratory at a university hospital. SUBJECTS: Eight healthy swine under volume- controlled mechanical ventilation. INTERVENTIONS: Flow probes were implanted in eight swine in order to continuously measure blood flow in the pulmonary artery and inferior vena cava. After a recovery phase of 14 days, the swine were subjected to various PEEP stages (0, 5, 10 cm H_2O) during volume-controlled positive pressure ventilation. MEASUREMENTS AND MAIN RESULTS: Continuous flow measurement took place in the pulmonary artery and inferior vena cava. Data on standard hemodynamic parameters were additionally acquired. Respiration-phase-specific analysis of right ventricular cardiac output and of additional hemodynamic function parameters followed, after calculation of mean values throughout five respiration cycles. PEEP at 5 cm H_2O led to significant decreases in inferior vena cava flow (4.1%), and in right ventricular cardiac output (5.2%); the respective decreases at PEEP 10 cm H_2O were 13.9% and 18.3%. In the inspiration phase at PEEP 10 cm H_2O, results revealed an overproportionally pronounced decrease in comparison with the expiration phase in inferior vena cava flow (-24.6% vs. -10%) and right ventricular cardiac output (-35% vs. -13.5%). This phenomenon is presumably caused by a PEEP-related increase in mean airway pressure by the amount of 10.7 cm H_2O in inspiration. CONCLUSIONS: Increases in PEEP during volume-controlled mechanical ventilation leads to respiration-phase-specific reduction of right ventricular cardiac output, with a significantly pronounced decrease during the inspiration phase. This decrease in cardiac output should be taken into particular consideration for patients with already critically reduced cardiac output.

The Pediatric Risk of Mortality (PRISM) Score and Injury Severity Score (ISS) for Predicting Resource Utilization and Outcome of Intensive Care in Pediatric Trauma—Castello FV, Cassano A, Gregory P, Hammond J. Crit Care Med 1999 May;27(5):985-988.

OBJECTIVE: Mortality prediction in trauma is assessed using the Injury Severity Score (ISS) and Revised Trauma Score using Trauma Injury Severity Score (TRISS) methodology. The Pediatric Risk of Mortality (PRISM) score assesses mortality risk in critically ill children. We compared the ability of PRISM and ISS (using TRISS methodology) to predict resource utilization and outcome in pediatric trauma. DESIGN: Retrospective chart and database review. SETTING: Pediatric intensive care unit (PICU). PATIENTS: Consecutive admissions to a PICU over a 2-yr period. MEASUREMENTS AND MAIN RESULTS: Demographic data including PICU resource utilization and outcome were recorded. Data were recorded on 1,052 admissions (31 deaths), including 125 pediatric trauma patients (11 deaths). Patients were stratified into low- and high-risk categories based on PRISM and ISS scores. Patients with PRISM scores <6 and ISS scores <10 were classified as low risk. While both low-risk PRISM and ISS scores readily identified survivors, PRISM was the more sensitive indicator of resource utilization. PRISM, however, performed less well in determining risk-adjusted mortality as compared with ISS. CONCLUSION: PRISM readily stratifies pediatric trauma patients for resource utilization. PRISM appears to underestimate mortality in pediatric trauma as compared with ISS using TRISS methodology.

Low-Dose Inhaled Nitric Oxide Improves the Oxygenation and Ventilation of Infants and Children with Acute, Hypoxemic Respiratory Failure—Ream RS, Hauver JF, Lynch RE, Kountzman B, Gale GB, Mink RB. Crit Care Med 1999 May;27(5):989-996.

OBJECTIVE: To describe the effects of inhaled nitric oxide on oxygenation and ventilation in patients with acute, hypoxic respiratory failure and to characterize those who respond to low doses with a significant improvement in P_{aO_2}. DESIGN: Prospective dose response trial of inhaled nitric oxide. Patients who demonstrated a ≥15% improvement in P_{aO_2} were randomized to receive conventional mechanical ventilation with or without prolonged inhaled nitric oxide. SETTING: Pediatric intensive care unit of a tertiary care children's hospital serving as a regional referral center for respiratory failure. PATIENTS: Pediatric patients with an acute parenchymal lung disease requiring mechanical ventilation, an F_{IO_2} of ≥0.5, a positive end-expiratory pressure of ≥7 cm H_2O, and whose P_{aO_2}/F_{IO_2} ratio was ≤ 160. INTERVENTIONS: P_{aO_2}, P_{aCO_2}, pH, heart rate, blood pressure, and methemoglobin were recorded at baseline and after inhaling 1, 5, 10, and 20 ppm of nitric oxide. Peak expiratory flow rate and mean airway resistance were measured while subjects received 0 and 20 ppm of inhaled nitric oxide. Patients were followed up until extubation or death. MEASUREMENTS AND MAIN RESULTS: Twenty-six patients (median age, 2.6 yrs [range, 1 mo-18.2 yrs]) were enrolled in the study. P_{aO_2} increased ($p < 0.001$) and P_{aCO_2} fell ($p<0.0001$) from baseline with the administration of inhaled nitric oxide. There was no statistical difference among 1, 5, 10, and 20 ppm with regard to effects on oxygenation. Sixteen patients (62%) responded to inhaled nitric oxide with a ≥15% improvement in P_{aO_2}; 14 of these responses occurred at a dose of 1 or 5 ppm. Response to inhaled nitric oxide was not associated with age, length of intubation, presence of primary lung disease, chest radiograph, or illness severity. Among patients weighing ≤20 kg, responders showed a greater fall in mean airway resistance ($p < 0.05$) than nonresponders. Mortality was not influenced by prolonged inhaled nitric oxide when analyzed by intention to treat. Patients receiving prolonged inhaled nitric oxide at doses of ≤20 ppm maintained methemoglobin levels of <3.0% and circuit concentrations of NO_2 of <1 ppm. CONCLUSIONS: Inhaled nitric oxide at doses of ≤5 ppm improves the oxygenation and (to a lesser extent) ventilation of most children with acute, hypoxic respiratory failure. The unpredictable response of patients necessitates individualized dosing of inhaled nitric oxide, starting at concentrations of ≤1 ppm. Inhaled nitric oxide at ≤20 ppm may exert a small salutary effect on bronchial tone. The benefits of prolonged inhaled nitric oxide remain unknown.

Preparing Hospitals for Toxicological Mass Casualties Events—Tur-Kaspa I, Lev EI, Hendler I, Siebner R, Shapira Y, Shemer J. Crit Care Med 1999 May:27(5):1004-1008.

OBJECTIVE: For most hospital staffs, treatment of chemical casualties presents an obscure and even frightening situation. We report our unique experience from hospital drills in order to improve hospital preparedness for patient management under mass casualty conditions involving hazardous chemicals. SETTING: Twenty-one major hospitals in Israel. INTERVEN-

TIONS: A unique hospital deployment plan for the management of chemical casualties was developed, and hospitals were required to have a full chemical practice drill every 3 to 5 yrs. These drills were designed as realistically as possible, and all included the use of personal protective equipment, decontamination, and treatment of simulated patients. Twenty-five percent of these patients, simulating children and adults, required intensive care and ventilation support. Hospitals were inspected and reviewed on the quality of treatment given and the overall continuity of care as well as on their administrative performance. RESULTS: Between 1986 to 1994, 30 full chemical practice drills were conducted in 21 major hospitals. Each drill included treatment of 100 to 400 simulated patients. The lessons from the hospital drills are described and were incorporated in the proposed revised hospital deployment plan. All hospitals significantly improved their ability to respond appropriately to these incidents. CONCLUSIONS: The level of preparedness for a chemical mass casualty scenario should be established according to the existing threat and the available resources. The proposed plan can serve as a basis for hospital planning and staff training worldwide, thus facilitating optimal care in the event of an incident involving toxic chemicals. A cost-effective scale for hospital preparation levels according to the existing threat is suggested.

Effect of Systemic Glucocorticoids on Exacerbations of Chronic Obstructive Pulmonary Disease. Department of Veterans Affairs Cooperative Study Group—Niewoehner DE, Erbland ML, Deupree RH, Collins D, Gross NJ, Light RW, et al. N Engl J Med 1999 Jun 24; 340(25):1941-1947.

BACKGROUND AND METHODS: Although their clinical efficacy is unclear and they may cause serious adverse effects, systemic glucocorticoids are a standard treatment for patients hospitalized with exacerbations of chronic obstructive pulmonary disease (COPD). We conducted a double-blind, randomized trial of systemic glucocorticoids (given for two or eight weeks) or placebo in addition to other therapies, for exacerbations of COPD. Most other care was standardized over the six-month period of follow-up. The primary end point was treatment failure, defined as death from any cause or the need for intubation and mechanical ventilation, readmission to the hospital for COPD, or intensification of drug therapy. RESULTS: Of 1840 potential study participants at 25 Veterans Affairs medical centers, 271 were eligible for participation and were enrolled; 80 received an eight-week course of glucocorticoid therapy, 80 received a two-week course, and 111 received placebo. About half the potential participants were ineligible because they had received systemic glucocorticoids in the pre-

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vious 30 days. Rates of treatment failure were significantly higher in the placebo group than in the two glucocorticoid groups combined at 30 days (33 percent vs. 23 percent, p=0.04) and at 90 days (48 percent vs. 37 percent, p=0.04). Systemic glucocorticoids (in both groups combined) were associated with a shorter initial hospital stay (8.5 days, vs. 9.7 days for placebo, p=0.03) and with a forced expiratory volume in one second that was about 0.10 liter higher than that in the placebo group by the first day after enrollment. Significant treatment benefits were no longer evident at six months. The eight-week regimen of therapy was not superior to the two-week regimen. The patients who received glucocorticoid therapy were more likely to have hyperglycemia requiring therapy than those who received placebo (15 percent vs. 4 percent, p=0.002). CONCLUSIONS: Treatment with systemic glucocorticoids results in moderate improvement in clinical outcomes among patients hospitalized for exacerbations of COPD. The maximal benefit is obtained during the first two weeks of therapy. Hyperglycemia of sufficient severity to warrant treatment is the most frequent complication.

Long-Term Treatment with Inhaled Budesonide in Persons with Mild Chronic Obstructive Pulmonary Disease Who Continue Smoking. European Respiratory Society Study on Chronic Obstructive Pulmonary Disease—Pauwels RA, Lofdahl CG, Laitinen LA, Schouten JP, Postma DS, Pride NB, Ohlsson SV. N Engl J Med 1999 Jun 24;340(25):1948-1953.

BACKGROUND: Although patients with chronic obstructive pulmonary disease (COPD) should stop smoking, some do not. In a double-blind, placebo-controlled study, we evaluated the effect of the inhaled glucocorticoid budesonide in patients with mild COPD who continued smoking. After a six-month run-in period, we randomly assigned 1277 subjects (mean age, 52 years; mean forced expiratory volume in one second [FEV_1], 77 percent of the predicted value; 73 percent men) to twice-daily treatment with 400 microg of budesonide or placebo, inhaled from a dry-powder inhaler, for three years. RESULTS: Of the 1277 subjects, 912 (71 percent) completed the study. Among these subjects, the median decline in the FEV_1 after the use of a bronchodilator over the three-year period was 140 mL in the budesonide group and 180 mL in the placebo group (p=0.05), or 4.3 percent and 5.3 percent of the predicted value, respectively. During the first six months of the study, the FEV_1 improved at the rate of 17 mL per year in the budesonide group, as compared with a decline of 81 mL per year in the placebo group (p<0.001). From nine months to the end of treatment, the FEV_1 declined at similar rates in the two groups (p=0.39). Ten percent of the subjects in the budesonide group and 4 percent of those in the placebo group had skin bruising (p<0.001). Newly diagnosed hypertension, bone fractures, postcapsular cataracts, myopathy, and diabetes occurred in less than 5 percent of the subjects, and the diagnoses were equally distributed between the groups. CONCLUSIONS: In patients with mild COPD who continue smoking, the use of inhaled budesonide is associated with a small one-time improvement in lung function but does not appreciably affect the long-term progressive decline.

Assessment of Usefulness of Endobronchial Ultrasonography in Determination of Depth of Tracheobronchial Tumor Invasion—Kurimoto N, Murayama M, Yoshioka S, Nishisaka T, Inai K, Dohi K. Chest 1999 Jun;115(6):1500-1506.

STUDY OBJECTIVE: We assessed the usefulness of endobronchial ultrasonography in the determination of the depth of tumor invasion of the tracheobronchial wall. METHODS: We performed a needle-puncture experiment on normal tissue of 45 specimens to determine the laminar structure of the tracheobronchial wall. In addition, we compared the ultrasonographic determinations of tumor invasion from 24 lung cancer cases with the histopathologic findings. RESULTS: The cartilaginous portions of the extrapulmonary bronchi and the intrapulmonary bronchi exhibited a five-layer structure. Starting on the luminal side, the first layer (hyperechoic) was a marginal echo, the second layer (hypoechoic) was the submucosal tissue, the third layer (hyperechoic) was the marginal echo on the inner side of the bronchial cartilage, the fourth layer (hypoechoic) was bronchial cartilage, and the fifth layer (hyperechoic) was the marginal echo on the outer side of the cartilage. In the membranous portions, the first layer (hyperechoic) was a marginal echo, the second layer (hypoechoic) was smooth muscle, and the third layer (hyperechoic) corresponded to the adventitia. Comparisons between the ultrasonograms and the histopathologic findings in 24 lung cancer cases revealed that depth diagnosis was the same in 23 lesions (95.8%) and was different in 1 lesion (4.2%). In the single case in which the findings were different, lymphocytic infiltration that protruded between the cartilage rings was mistakenly interpreted as tumor infiltration. CONCLUSIONS: This method allows visualization of the laminar structure of the tracheobronchial wall, which is impossible with other diagnostic imaging methods.

Combined Effects of a Mechanical Nasal Dilator and a Topical Decongestant on Nasal Airflow Resistance—Lorino AM, Lofaso F, Dahan E, Coste A, Harf A, Lorino H. Chest 1999 Jun;115(6):1514-1518.

The goal of this study was to compare the isolated and combined effects of two treatments being used to reduce nasal airflow resistance (NR): an internal nasal mechanical dilator (Nozovent; Prevancure; Ste Pouret, Paris, France) and a topical decongestant, fenoxazoline hydrochloride (Aturgyl; Synthelabo; Le Plessis-Robinson, France). The study was performed in 17 healthy subjects. NR was estimated by active posterior rhinometry at a 0.5 L/s flow under four conditions: in the basal state, with the internal nasal mechanical dilator, after treatment with fenoxazoline hydrochloride, and with both fenoxazoline hydrochloride and the mechanical dilator. The mean NR (± SD) decreased from 1.65±0.54 cm H_2O/L/s in the basal state to 1.02±0.27 cm H_2O/L/s with the mechanical dilator (p < 0.001), 1.03±0.47 cm H_2O/L/s with fenoxazoline hydrochloride (p < 0.001), and 0.48±0.15 cm H_2O/L/s with both the mechanical dilator and fenoxazoline hydrochloride (p < 0.001). The decreases in NR observed after using either the mechanical dilator ($\Delta NR(N)$) or fenoxazoline hydrochloride ($\Delta NR(A)$) were not significantly different. The decrease in NR observed with both ($\Delta NR(N + A)$) was not significantly different from the sum $\Delta NR(N) + \Delta NR(A)$: 1.16±0.53 cm H_2O/L/s vs 1.25±0.63 cm H_2O/L/s, respectively (p > 0.05). $\Delta NR(N + A)$ strongly correlated with $\Delta NR(N) + \Delta NR(A)$: $\Delta NR(N + A) = 0.80\ (\Delta NR(N) + \Delta NR(A)) + 0.15$ (r = 0.96; p < 0.0001). However, the slope of the regression line of $\Delta NR(N + A)$ vs $\Delta NR(N) + \Delta NR(A)$ was significantly lower than unity (p < 0.003). These results demonstrate that, although not totally additive, the effects of using the mechanical dilator and fenoxazoline hydrochloride are cumulative. Further studies that include patients with nasal obstruction would allow us to better evaluate the benefit of a therapy combining both treatments.

Bedside Evaluation of Efficient Airway Humidification During Mechanical Ventilation Of the Critically Ill—Ricard JD, Markowicz P, Djedaini K, Mier L, Coste F, Dreyfuss D. Chest 1999 Jun;115(6):1646-1652.

STUDY OBJECTIVE: To determine the correlation between simple rating of condensation seen in the flex-tube connecting the heating and humidifying device used with the endotracheal tube and hygrometric parameters (absolute and relative humidity and tracheal temperature) measured by psychrometry. DESIGN: Prospective randomized clinical trial. SETTING: Medical ICU of Louis Mourier Hospital, Colombes, France, a university-affiliated teaching hospital. PATIENTS: Forty-five consecutive mechanically ventilated critically ill patients. INTERVENTIONS: Patients undergoing mechanical ventilation were randomly assigned to receive humidification with one of the four heat and moisture exchangers (HMEs) tested or with a conventional heated humidifier. MEASUREMENTS: The hygrometric performances of four HMEs (BB2215, BB50, and BB100 from Pall

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Biomedical, Saint-Germaine-en-Laye, France; and Hygrobac-Dar from Mallinckrodt, Mirandola, Italy) and a heated humidifier (Fisher & Paykel; Auckland, New Zealand) were studied after 3 h and also after 48 h of use for the Hygrobac-Dar and correlated to a clinical visual inspection rating the amount of condensation in the flex-tube of the endotracheal tube. RESULTS: A total of 95 measurements in 45 patients were performed. The best hygrometric parameters were obtained with the heated humidifier ($p < 0.001$). The Hygrobac-Dar yielded significantly higher values for both humidities and tracheal temperature than the other three HMEs ($p < 0.001$). The performance of Hygrobac-Dar was unchanged after 48 h of use. There was a significant correlation between the condensation seen in the flex-tube and the hygrometric parameters measured by psychrometry (absolute humidity, rho = 0.7; relative humidity, rho = 0.7; tracheal temperature, rho = 0.5, $p < 0.0001$). CONCLUSION: In mechanically ventilated ICU patients, visual evaluation of the condensation in the flex-tube provides an estimation of the heating and humidifying efficacy of the heating and humidifying device used, thus allowing the clinician bedside monitoring of airway humidification.

A Comparison of Bronchodilator Therapy Delivered by Nebulization and Metered-Dose Inhaler in Mechanically Ventilated Patients—Marik P, Hogan J, Krikorian J. Chest 1999 Jun;115(6):1653-1657.

BACKGROUND: The optimal method of delivering bronchodilators in mechanically ventilated patients is unclear. The purpose of this study was to compare the pulmonary bioavailability of albuterol delivered by the nebulizer, the metered-dose inhaler (MDI) and spacer, and the right-angle MDI adaptor in ventilated patients using urinary analysis of drug levels. METHODS: Mechanically ventilated patients who had not received a bronchodilator in the previous 48 h and who had normal renal function were randomized to receive the following: (1) five puffs (450 microg) of albuterol delivered by the MDI with a small volume spacer; (2) five puffs of albuterol delivered by the MDI port on a right-angle adaptor; or (3) 2.5 mg albuterol delivered by a nebulizer. Urine was collected 6 h after the administration of the drug, and the amounts of albuterol and its sulfate conjugate were determined in the urine by a chromatographic assay. RESULTS: Thirty patients were studied, 10 in each group: their mean age and serum creatinine level were 62 years and 1.3 mg/dL, respectively. With the MDI and spacer, (mean ± SD) 169±129 microg albuterol (38%) was recovered in the urine; with the nebulizer, 409±515 microg albuterol (16%) was recovered in the urine; and with the MDI port on the right-angle adaptor, 41±61 microg albuterol (9%) was recovered in the urine ($p = 0.02$ between groups). The level of albuterol in the urine was below the level of detection in four patients in whom the drug was delivered using the right-angle MDI adaptor. CONCLUSION: The three delivery systems varied markedly in their efficiency of drug delivery to the lung. As previous studies have confirmed, this study has demonstrated that using an MDI and spacer is an efficient method for delivering inhaled bronchodilators to the lung. The pulmonary bioavailability was poor with the right-angle MDI port. This port should not be used to deliver bronchodilators in mechanically ventilated patients.

Effect of Combined Kinetic Therapy and Percussion Therapy on the Resolution of Atelectasis in Critically Ill Patients—Raoof S, Chowdhrey N, Raoof S, Feuerman M, King A, Sriraman R, Khan FA. Chest 1999 Jun;115(6):1658-1666.

BACKGROUND: Some critically ill patients have difficulty in mobilizing their respiratory secretions. These patients can develop pulmonary atelectasis that may result in hypoxemia. There are some data to show that atelectasis may be prevented by turning a patient from side to side utilizing special beds. STUDY OBJECTIVES: To determine the role of kinetic therapy (KT) combined with mechanical percussion (P) in the resolution of established atelectasis of the lungs and hypoxemia in critically ill, hospitalized patients. (KT was defined as rotation of a patient along the longitudinal axis of ≥ 40 degrees to each side continuously.) DESIGN: Prospective and randomized study (2:1 test to control group). PATIENTS: Twenty-four patients with respiratory failure, either mechanically ventilated or spontaneously breathing, who demonstrated segmental, lobar, or unilateral entire lung atelectasis were studied. SETTING: Medical ICU and adult respiratory ward in a county hospital in New York. INTERVENTIONS: Seventeen patients were treated with KT combined with mechanical P using a KT system (Triadyne Kinetic Therapy System; KCI; San Antonio, TX). Seven patients received manual repositioning and manual P every 2 h. Both groups received similar conventional therapy with inhaled bronchodilators and suctioning. RESULTS: Partial or complete resolution of atelectasis was seen in 14 of 17 patients (82.3%) in the test group as compared with 1 of 7 patient (14.3%) in the control group. The median duration to resolution of atelectasis was 4 days in the test group. Bronchoscopy was performed in 3 of 7 patients in the control group, but in none of the patients in the test group. A cost of $720 was incurred per patient for utilizing the specialty beds for a mean duration of 4 days. An improvement in oxygenation index occurred in the test group (change in baseline P_{aO_2}/fraction of inspired oxygen from 207.4±106.7 mm Hg to 318±100.7 mm Hg) at the end of therapy, while the control group showed a reduction over a similar duration of time (181.3±96.3 mm Hg to 112±21.2 mm Hg). CONCLUSIONS: KT and mechanical P therapy resulted in significantly greater partial or complete resolution of atelectasis as compared with conventional therapy. There was a generalized trend toward statistical significance in the improvement of oxygenation and a reduced need for bronchoscopy in the group receiving KT and P therapy.

Reliability of Maximal Respiratory Pressures in Multiple Sclerosis—Smeltzer SC, Lavietes MH. Chest 1999 Jun;115(6):1546-1552.

OBJECTIVE: To assess the reliability of maximal inspiratory pressure ($P_I$max) and maximal expiratory pressure ($P_E$max) in subjects with multiple sclerosis (MS) and healthy control subjects by identifying the number of testing sessions and the number of measurements needed in a single testing session to obtain consistent, reproducible results. DESIGN: A descriptive, comparative design with repeated measures was used. SETTING: Four sets of 10 $P_I$max and 10 $P_E$max measurements were obtained over a 4-week period from MS subjects in their homes. The same measurements were obtained from healthy control subjects in a private setting. SUBJECTS: Seventy-two MS patients and 61 healthy control subjects participated in the study. MEASUREMENT: $P_I$max and $P_E$max values were obtained by using previously published methods. RESULTS: Mean $P_E$max and $P_I$max values for MS patients differed over the first three of the four testing sessions. By contrast, mean $P_E$max and $P_I$max values for healthy control subjects differed only when the first session values were compared with values from the last three sessions. For MS patients, $P_E$max and $P_I$max increased between the first and 10th trial during the first testing session, but not during the subsequent three sessions. CONCLUSIONS: The results of this study suggest that several practice sessions should be provided in order to obtain reliable $P_E$max and $P_I$max values in persons with MS. At least one practice session should be provided for healthy control subjects before identifying a baseline.

Thoracic Imaging in the Intensive Care Unit: Improving Clinical Skills and Access Means Better Patient Care

Patricia A Doorley RRT and Charles G Durbin Jr MD

As the intensity and cost of medical care continues to increase, respiratory therapists and other patient care providers are expected to assume a more proactive role in evaluation of patients and management of their diseases. Respiratory care providers in particular have a responsibility to assess patient changes and evaluate the clinical responses to expensive and time-consuming therapies. Along with physical assessment, diagnostic radiography can be very helpful in guiding our management of patients and ensuring positive and cost-effective outcomes.

One of the most useful clinical assessment tools in an intensive care unit (ICU) is the chest radiograph. The technological advances and analytical approaches described in this issue's series of papers, "Thoracic Imaging in the ICU," will help bedside clinicians incorporate examination of chest and other radiographs into their patient assessment repertoire. While most clinicians' radiologic skills will not replace film evaluation by a formally trained radiologist, there are certain aspects of the radiologic exam to which the bedside clinician can make important contributions. Experienced radiologists often identify subtle but important abnormalities; caregivers at the bedside know where to look for the larger and more imperative concerns. Because of their knowledge of current clinical information and their awareness of physical examination abnormalities, inspection of the radiograph is much more likely to be helpful to bedside providers. For example daily variations in radiographic image quality, which are expected because of the nature of ICU care, are easier to interpret if the same person is viewing the images and the patient on a daily basis. Changes in densities of the lung fields secondary to a change in fluid status can more readily be understood—and therapy appropriately adjusted—by an individual who is also aware that there are rales on chest exam and that the wedge pressure is 24 mm Hg.[1]

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Studies examining the impact of routine radiographs on patient outcome have shown significant delay in identification and treatment of easily identified pathology such as endotracheal tube misplacement, new infiltrates, lung collapse, and pneumothorax. While as many as 40–60% of "routine" radiographs show these easily recognizable and potentially harmful situations, a delay of as many as 6 hours is not unusual, because of the failure of the "system" to notify the responsible clinician. A solution is to have the caregiver seek out and examine the film immediately. The digitization, electronic storage, and timely transmission of radiographic images have improved the availability of these images so that they can now be inspected at the patient's bedside.[2] With 20–30% of films demonstrating abnormalities requiring change of treatment, respiratory therapists and other bedside caregivers have the responsibility to systematically examine these studies. This becomes especially important in a time of limited resources, when moving patients seamlessly through the ICU is a fiscal necessity.[3–5]

The papers in this issue of *Respiratory Care* deal with both simple and complicated radiographic techniques and diagnoses. The intent is not to make every respiratory therapist a radiologic expert. However, respiratory care providers can and should become adept at identifying simple but important problems. Every respiratory care provider should be able to recognize pneumothorax, lung collapse, mediastinal shift, fluid in the pleural space, endotracheal tube displacement, and vascular line misplacement. Most respiratory therapists will improve their diagnostic skills with this review.

Just as no one doubts the value of a stethoscope in the completion of a thorough lung examination, most intensivists believe the radiograph is more sensitive and specific for making or confirming the most important pulmonary diagnoses. Not surprisingly, radiographs obtained after an invasive procedure demonstrate the highest incidence of treatable conditions. Through the use of thoracic imaging, clinicians can uncover important clues to explain the patient's failure to improve with appropriate therapy, and thereby enhance patient care. It is our hope that the fol-

lowing introductory papers will stimulate our readers to take a more aggressive approach to radiograph analysis and incorporate personal evaluation of chest radiographs and other radiographs into their routine patient assessment.

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Thoracic Imaging in the Intensive Care Unit

There have been remarkable advances in intensive care, technologic support, monitoring, and diagnostic devices and tools. One of these tools is the ubiquitous chest radiograph. In this computer age, the chest radiograph is often part of a department or hospital-wide move to the use of computed radiography. Computed radiography provides much more consistency from exam to exam, and is a steppingstone to a fully integrated picture archive system. This increased access to radiologic images makes it increasingly important for many different health care providers to have a basic understanding of the chest radiograph as it pertains to the intensive care patient.

Patients in the hospitals are more ill than ever before. With the emphasis on cost savings, routine medical care can be provided on an outpatient basis for short hospital stays. However, severely injured or ill patients tend to be even sicker for longer periods than in the past. Those very ill or injured patients who might have died are now kept alive thanks to modern critical, emergency, and surgical care. The traditional "intensive" care unit has, like everything else in medicine, undergone subspecialization. Larger hospitals now have separate intensive care units for respiratory, surgical, cardiac, pediatric, and other types of patients, each with dedicated subspecialty providers (nurses, doctors, respiratory therapists, and other support staff).

The authors of this series of articles were solicited from the presenters of a symposium on critical care imaging presented at the meeting of the Society of Thoracic Radiology, March 8–12, 1998, in San Juan, Puerto Rico. The authors are nationally recognized authorities in thoracic imaging, and are on faculty at major university hospital tertiary care referral centers. The articles presented are meant to cover the spectrum of modern day intensive hospital care from an imaging perspective.

As the editors of this series we have tried to keep overlap to a minimum. Each article can be read individually, focusing on a particular work situation; however, the articles are for the most part complementary.

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Pitfalls In Portable Chest Radiology

Heber MacMahon MD

Introduction
Technical Considerations
Equipment
Beam Projection
Lung Inflation and Patient Posture
Effect of Exposure Variations
Projection Variations
Atypical Distribution of Pleural Effusion
Unusual Air Collections
Atelectasis
Digital Radiography
Summary

[Respir Care 1999;44(9):1018–1032] *Key words: portable radiography, x-ray interpretation, radiography artifacts, digital radiography, radiologic technique.*

Introduction

Portable (bedside) radiography accounts for an increasing proportion of all chest x-ray examinations performed in hospitals as the emphasis has shifted toward critical care and the use of cardiopulmonary monitoring devices has become commonplace. In several large medical centers in the United States, 40–50% of all chest radiographs are now performed at the bedside with mobile equipment.[1] In spite of increased utilization, the quality of bedside radiography is highly variable, and the average quality is generally poor compared to that of examinations performed with fixed equipment in the radiology department. This makes portable radiographs more difficult to interpret, while variations in technique and projection from day to day compound the problem. Many misdiagnoses are attributable to poor image quality as well as unfamiliarity with the atypical appearance of certain types of pathology on semierect bedside examinations.

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Technical Considerations

Portable radiographs differ from standard upright chest radiographs in several respects, related to equipment, projection, and patient posture.

Equipment

Mobile radiography machines have advanced to the point where their output is no longer a limiting factor in the quality and consistency of radiographs. Selection of x-ray factors (kilovolts peak [kVp], milliampere-second [mAs]) can be based on issues other than constraints of the equipment. On the other hand, significant limitations exist at the receptor, because it is not mechanically or electrically linked to the x-ray source. As a result, exposure is not automatically controlled, and day-to-day variations in film density are commonplace.

Even when exposure is optimal or when digital systems are used that compensate for exposure variations, portable chest radiographs are typically inferior in quality to standard upright examinations. This is principally due to the fact that antiscatter grids are not routinely used in adult portable chest radiography. In one survey in the United States, only 12% of institutions reported using antiscatter grids for adult portable chest radiography.[1]

Failure to use a grid results in large amounts of scattered radiation impinging on the detector, with resulting loss of contrast and degradation of diagnostic quality.[2] Antiscatter grids are routinely used in the x-ray department for adult patients, and the resulting improvement in image quality is substantial, especially in larger patients. One objection to the routine use of grids is their weight and bulk, compared to a standard film cassette. Another concern has been the difficulty of obtaining accurate grid alignment in the bedside situation. This is an important issue, because a badly misaligned grid can provide results worse than nongrid technique. Fortunately, solutions to these problems are being developed, and the use of grids for portable work is increasing as radiologists and vendors have become more aware of the advantages of using grids in bedside work.[1] Therefore, several factors contribute to variations in film density and quality, which can affect interpretation.

Digital systems are increasingly being used for bedside work, mainly because they can compensate for exposure errors and thus improve the consistency of image quality and reduce the repeat rate.[3] Nonetheless, some digital systems are still susceptible to density and contrast variations due to changes in patient positioning and collimation. The photostimulable phosphor plates that are currently used for digital radiography are more sensitive to scatter than screen-film systems, and, therefore, use of an antiscatter grid is particularly important. Unfortunately, the image processing software cannot compensate adequately for the effects of grid misalignment. Density and contrast can be corrected to some extent, but unacceptably high levels of noise may result when the grid is not correctly aligned with the central x-ray beam.

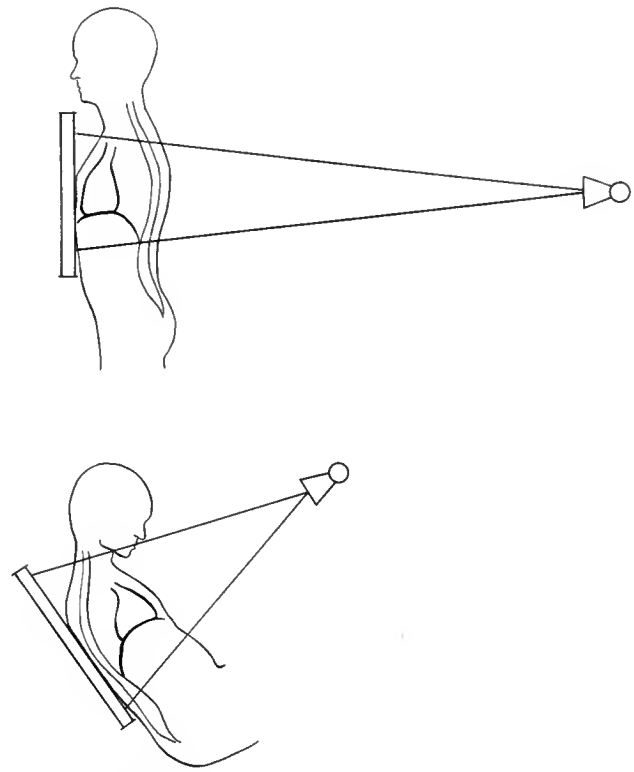

Fig. 1. Schematic of standard erect posteroanterior (PA) and portable anteroposterior (AP) chest radiography. In the case of the PA radiograph, the patient is standing upright, and the x-ray source is 72 inches from the image detector, so that magnification due to beam divergence is minimal. The central x-ray beam is horizontal, which facilitates detection of air-fluid levels or subdiaphragmatic free air. Portable (bedside) radiography is generally performed with the patient semi-erect, as shown, or supine. Due to the AP projection and closer x-ray source, cardiac magnification is increased. Because the central x-ray beam is not horizontal, air-fluid levels or pneumoperitoneum may be difficult to detect.

Beam Projection

For standard erect chest radiographs, the central x-ray beam is horizontal, but with portable chest x-ray the beam is almost never horizontal. This is arguably the single most important source of interpretative pitfalls in portable radiography. In practice, the x-ray beam angle is determined by the position of the patient. If the patient is reclining at a semi-erect angle of 45°, the film cassette will be placed behind the patient at the same angle, and the x-ray beam will be angled downward at approximately 90° to the cassette (45° from horizontal). As a result, air-fluid levels will not be visible and small air collections that are oriented horizontally (such as pneumoperitoneum) will be difficult to see in the radiograph (Fig. 1).

The source-to-image-receptor distance (formerly called the focus-film distance or FFD), is also different in portable radiography. A 72-inch (180 cm) distance is used for standard upright radiography in the radiology department. This produces minimal magnification because of beam divergence, and the posteroanterior patient position further minimizes any effects on cardiac size. For portable examinations, the distance is not fixed, but it is usually considerably less than 72 inches. Thus, there is greater beam divergence effect. Cardiac magnification is compounded by the anteroposterior (AP) portable projection that places the heart at a greater distance from the receptor.

A second, less well understood effect of projection relates to variations in beam centering and angulation relative to the patient's anatomy. While the appearance caused by patient rotation is widely recognized, alterations caused by vertical beam angulation are seldom appreciated. For example, if a portable radiograph is obtained from a lordotic projection on one occasion and from an antilordotic projection on another occasion, the effects on the appearance of pleural effusions, basal consolidation, and the mediastinal silhouette can be profound, often leading to a diagnosis of interval change where in fact none exists.

Fig. 2. Unilateral grid cutoff. In this portable radiograph the grid is misaligned, at an angle to the central x-ray beam, resulting in underpenetration of one side of the radiograph. Note that the opacity extends beyond the margins of the lung, and therefore, this should not be misinterpreted as a pulmonary abnormality.

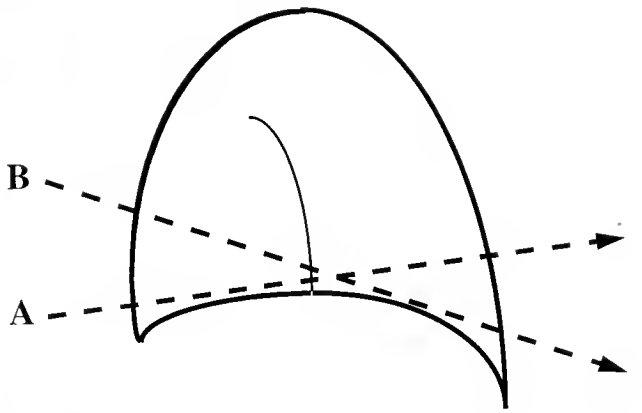

Fig. 3. Schematic depiction of vertical variation in x-ray beam projection, from a lateral perspective. Note that a more lordotic projection (A) results in visualization of a more anterior part of the diaphragm than a more caudal projection (B). Such variations can substantially affect the appearance of basilar disease.

Lung Inflation and Patient Posture

Radiologists and intensivists are well aware that the degree of pulmonary inflation can alter the appearance of the lungs and can minimize or accentuate the severity of diffuse disease. Still, in a clinical setting, there is a tendency to underestimate these effects. Similarly, alterations in ventilator settings in patients receiving ventilatory assistance can markedly alter the radiographic appearance.

In portable chest radiography a truly erect patient position is almost never obtained. So-called erect portable examinations are usually performed with the patient in a semi-erect position. Often, a supine position is used. In fact, a supine position is more conducive to good lung expansion than a doubled-forward, semi-erect posture. In either case, the patient's position has important effects on the distribution of abnormal air and fluid collections, notably pleural effusions and pneumothorax. As a result, the appearance may change dramatically from one examination to the next if positioning is not consistent.

Effect of Exposure Variations

Many patients in intensive care units (ICUs) have diffuse lung disease such as acute respiratory distress syndrome or cardiogenic pulmonary edema. Daily chest radiographs obtained to detect interval change must be interpreted with caution when variations in technique, lung expansion, or projection are present. Accurate interpretation of diffuse disease requires consistent radiographic technique and awareness of factors that can alter the radiographic appearance of the lungs.

Increased severity of pulmonary disease is associated with increased opacity, and often with decreased inflation. Unfortunately, lung opacity will vary with technique, and inflation can be affected by many factors, including ventilator settings, exposure timing, and voluntary effort. To minimize variations in technique, kVp and distance should be standardized. Exposure should be varied for individual patients by altering the mAs (tube current and exposure time), and a record of previous exposures should be used as a guide for subsequent examinations. If possible, a digital radiography system should be used. Apart from their well-known advantages in reducing the need for repeat examinations due to exposure errors, digital systems produce images that are much more consistent than traditional screen-film systems, particularly in terms of density and contrast. Ideally, ventilator settings should be recorded and changes noted. Serial radiographs, including multiple previous images when available, should be viewed side by side so that trends can be detected, and so that technical variations can be more readily appreciated. This practice is critically important in distinguishing actual from spurious changes in diffuse lung disease.

Another interpretive pitfall can arise from grid misalignment. If the central x-ray beam is not correctly aligned with the center of the grid, asymmetric "grid cutoff" can occur. This means that the primary radiation is being partially blocked by the lead strips of the grid.[4] When the grid is oriented vertically, unilateral cutoff may occur toward one side of the radiograph, resulting in apparently increased

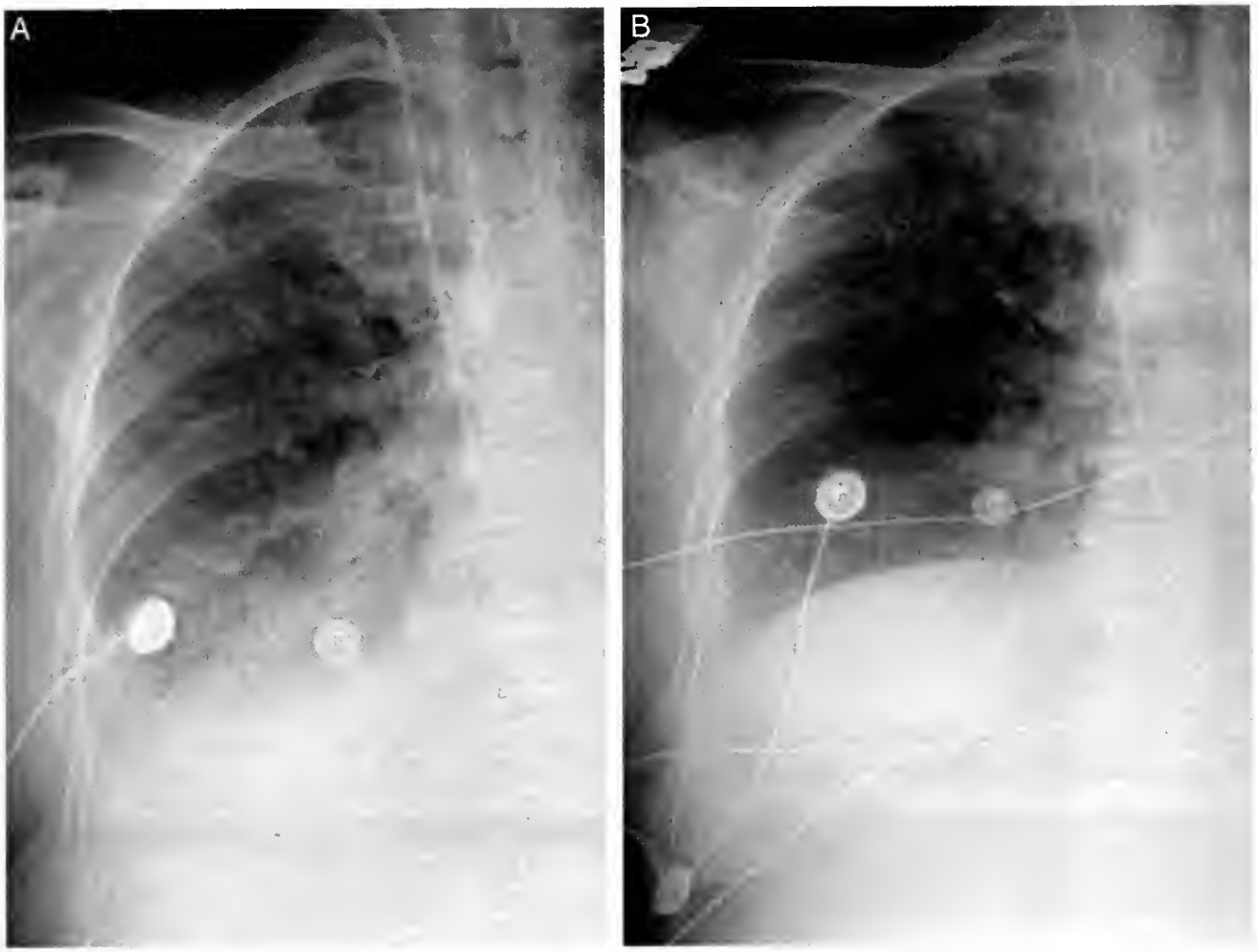

Fig. 4. A: Portable chest radiograph of a patient with a right pleural effusion shows obliteration of the diaphragmatic silhouette. B: A portable radiograph obtained the next day with a more lordotic projection shows a sharply defined diaphragm. This change is consistent with the different perspective of the lordotic projection, as illustrated in Figure 3, and should not be misinterpreted as indicating an actual change in the extent of disease.

opacity over one lung (Fig. 2). This is commonly misinterpreted as unilateral pulmonary or pleural disease. The key to avoiding this particular pitfall is to analyze the extent of any apparent unilateral opacity on the radiograph. If the opacity extends beyond the margins of the lung, it should not be attributed to pulmonary or pleural disease. While unilateral grid cutoff is not confined to portable applications, it is considerably more common in this situation.

Projection Variations

Variations in x-ray beam projection are another common source of errors in interpretation. In a standard upright chest radiograph the position of the patient tends to be more or less consistent from one examination to the next, because of the fixed nature of the equipment and the requirement for a standing position. In the case of a portable chest radiograph, however, both the position of the patient and the position of the x-ray tube can vary over a wide range. Rotation of the patient relative to the x-ray source and receptor can produce profound effects on the appearance of the mediastinum. Such rotation can be misinterpreted as pathological mediastinal widening, particularly in an older patient with a tortuous aorta. More experienced clinicians and radiologists are generally aware of this phenomenon. On the other hand, the effects of vertical variations in beam projection are less well understood, and the effects of variations in vertical beam projection are frequently misinterpreted as pathological change when in fact no real change exists. Fig. 3 illustrates the basis of this phenomenon. Note that in this schematic example, when the x-ray tube is aligned with the inferior part of the thorax and the x-ray beam has a lordotic projection, the anterior part of the diaphragm is tangential to the beam. But when

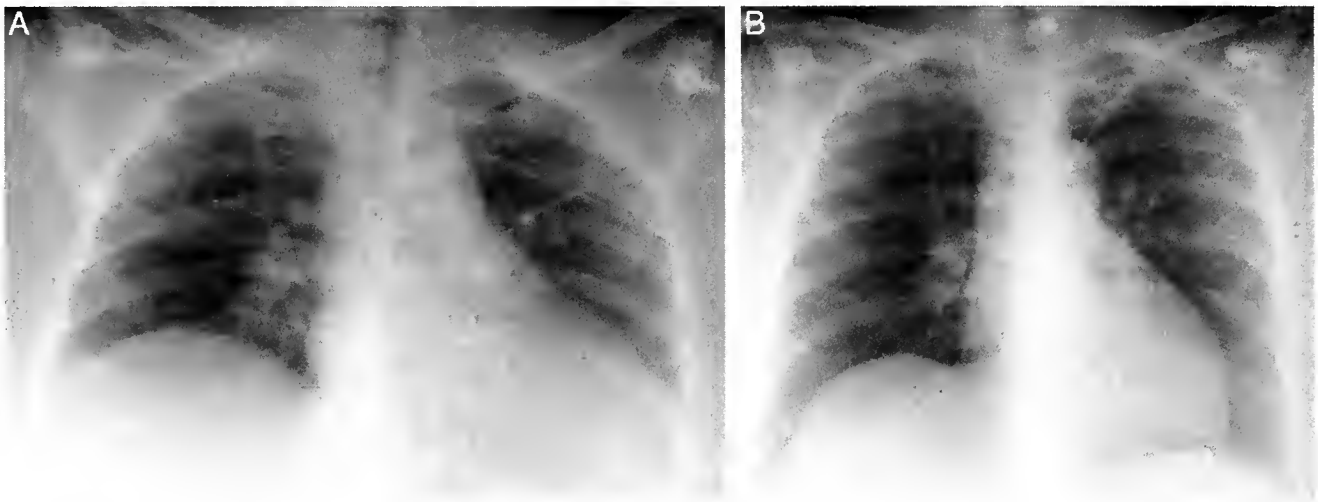

Fig. 5. A: Underinflated lungs on a portable radiograph of an obese patient produce an appearance suggestive of mild congestive heart failure. B: A repeat portable radiograph of the same patient obtained only minutes later, shows markedly improved pulmonary inflation, and reduced magnification, because of better positioning. There is no evidence of cardiopulmonary disease.

the x-ray tube is centered higher and the beam is directed more caudally, the beam will be tangential with a more posterior part of the diaphragm. In the case of a patient with posteriorly located pulmonary consolidation or pleural effusion, this type of projection variation can make the difference between a sharply defined diaphragmatic outline and one that is completely obliterated, without any actual change in the patient's condition (Fig. 4).

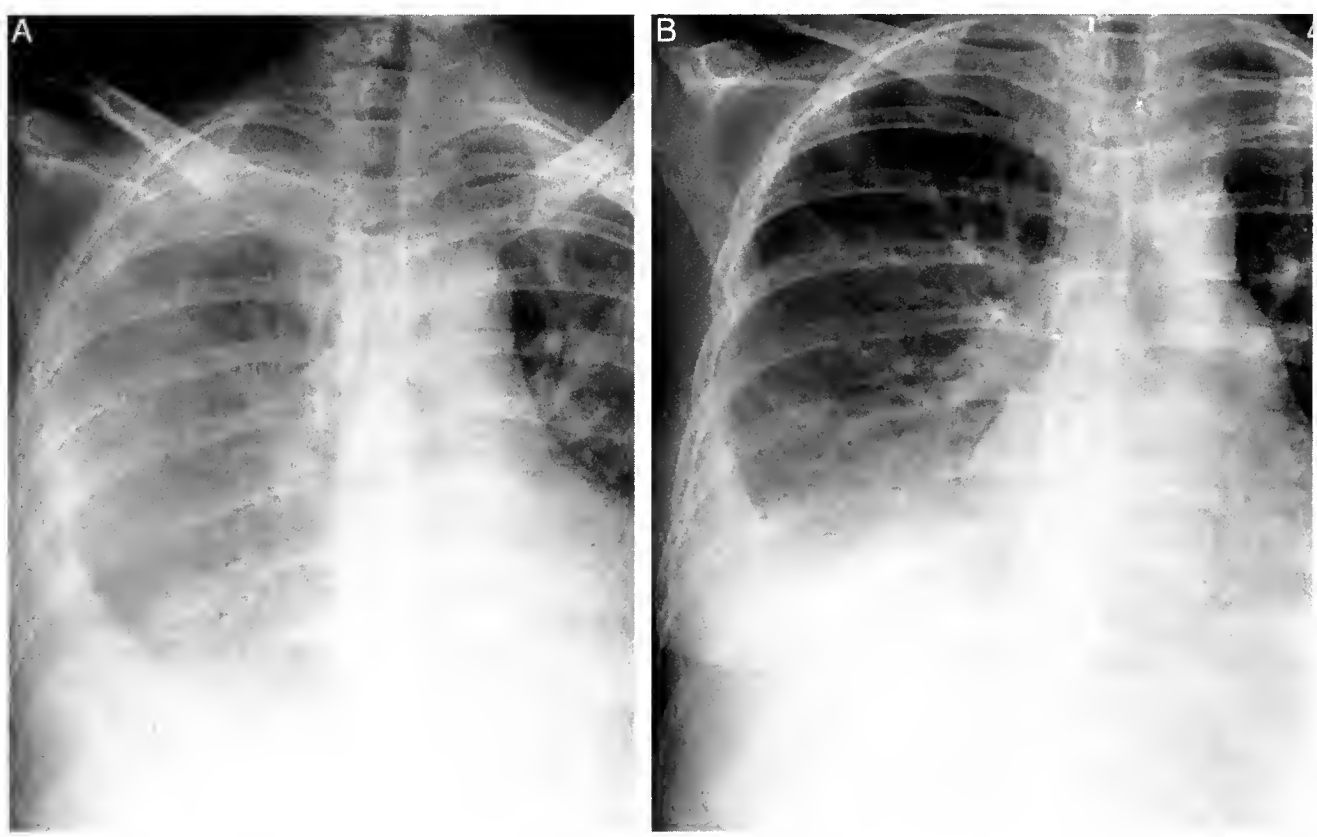

Fig. 6. A: Supine chest radiograph shows layering out of a large pleural effusion that extends over the lung apex. B: A radiograph of the same patient obtained with a semi-erect posture produces a different appearance because of basilar accumulation of the pleural fluid.

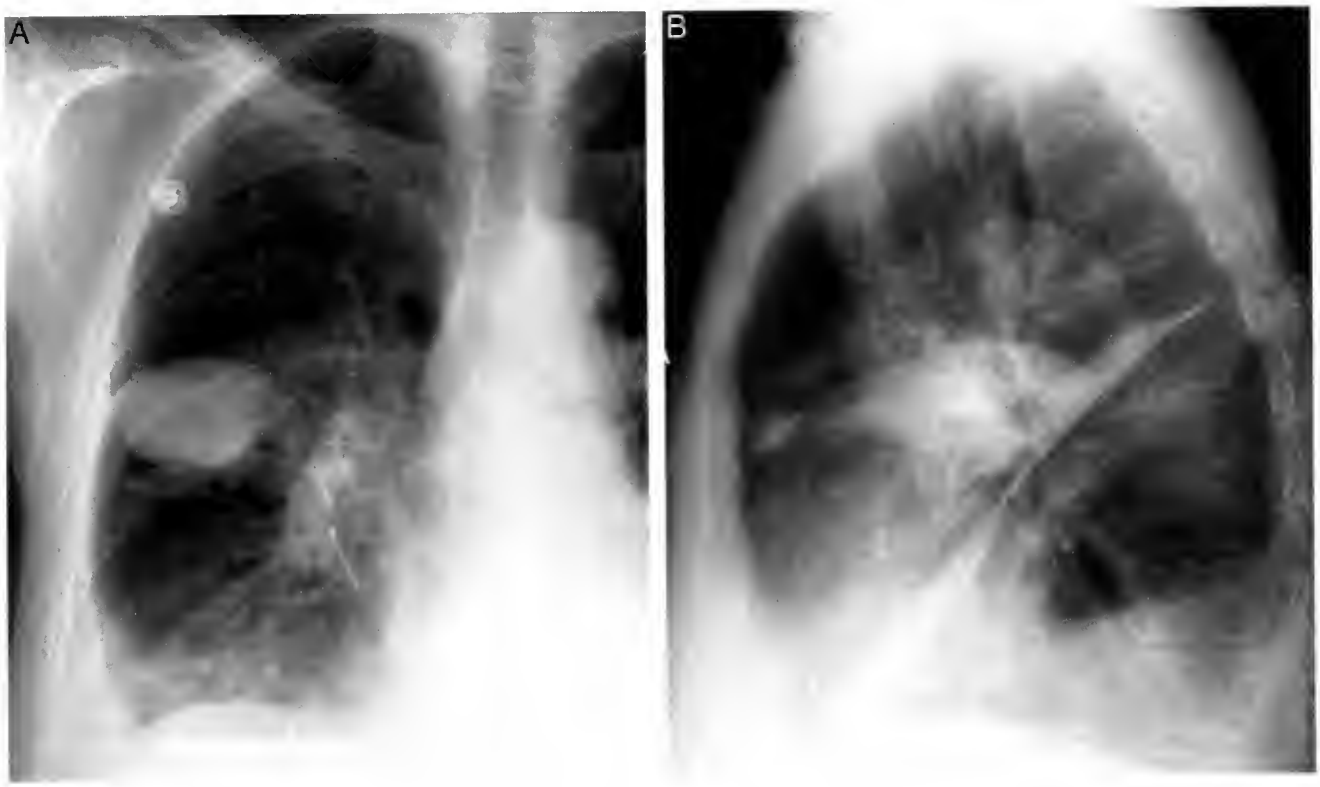

Fig. 7. A: "Pseudotumor" due to pleural fluid loculated in the minor fissure. B: A lateral view clearly demonstrates the relationship of the "mass" to the fissure, suggesting its true nature.

In the case of a series of chest radiographs, any apparent change at the lung bases should be correlated with changes in vertical beam projection, as indicated by the number of ribs projected above the clavicle. To some extent, this phenomenon also occurs in the case of upright standard chest radiographs, which may be obtained in a posteroanterior or AP projection (Fig. 5). The AP projection tends to be more lordotic, commonly resulting in obliteration of the silhouette of the medial part of the left hemidiaphragm by the heart. This should not be misinterpreted as pathological change. When variations in projection are combined with differences in pulmonary inflation, the effects on the appearance of the chest can be dramatic—and potentially misleading.

Underinflation of the lungs is particularly common on portable chest radiographs because of patient posture, increased upward pressure on the diaphragm in the semi-erect sitting position, and, frequently, patient inability to fully cooperate. The combination of underinflation with obesity can produce an appearance that is often misinterpreted as mild interstitial edema (see Fig. 5). As a general rule, merely hazy opacity and indistinctness of pulmonary vessels on a portable radiograph are unreliable criteria for diagnosing pulmonary edema. Even mild interstitial edema produces thickening of pleural fissures due to subpleural edema, septal lines (Kerly B lines), and small amounts of pleural fluid.

Atypical Distribution of Pleural Effusion

The appearance of a pleural effusion on a portable chest radiograph depends on the patient's position and the beam projection. Variations in patient position between supine and semi-erect make it difficult to determine changes in the size of the effusions on serial portable chest radiographs (Fig. 6). In a completely supine position, the pleural effusion will layer out posteriorly and, if large enough, can extend over the lung apex and medial to the upper lobe, producing apparent mediastinal widening. In the typical semi-erect patient position of portable chest radiography, pleural effusions preferentially collect in the posterior costophrenic angles, and their presence can be inferred, on good quality radiographs, from increased retrodiaphragmatic opacity. Blunting of the medial and lateral costophrenic angles generally occurs only after several hundred milliliters of fluid have accumulated. Because of the large effects of patient position and x-ray beam projection on the appearance of effusions, apparent interval changes in the volume of pleural effusions should be interpreted with caution.

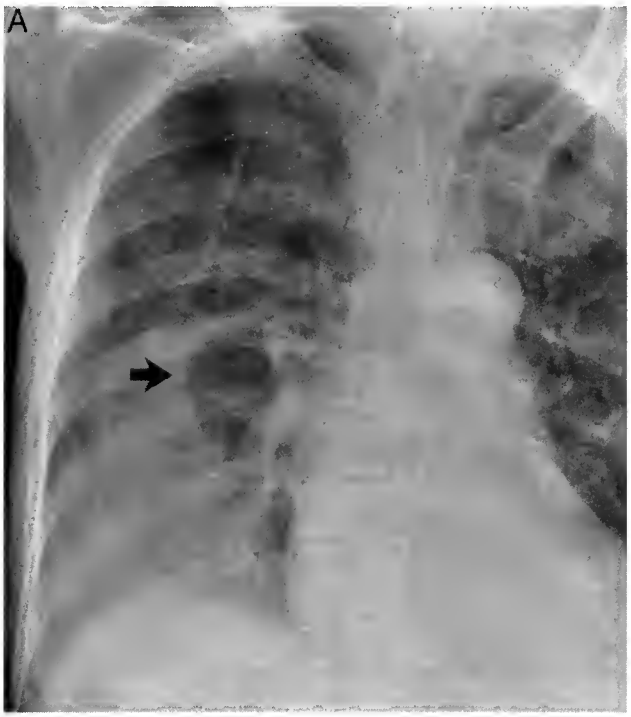

Fig. 8. A: A clearly defined radiolucent area (arrow) is visible on this semi-erect portable chest radiograph of a patient with fluid overload and a right pleural effusion. This appearance, which can be misinterpreted as a cavity, is typical of pleural fluid extending into the major fissure, and outlining the superior segment of the lower lobe. B: A computed tomography scan of another patient with a pleural effusion illustrates how pleural fluid can extend into the major fissure and outline the partially collapsed superior segment of the right lower lobe (arrow), which produces a focal area of radiolucency, as shown in Figure 8A.

When pleural fluid becomes loculated in the pleural fissures, it can present as a discrete mass-like opacity that can simulate a tumor (Fig. 7). These "pseudotumors" necessarily coincide with the location of the fissures, and often have a slightly elliptical configuration, with the long axis corresponding to the direction of the fissure. Such loculation is observed most commonly with resolving effusions. A lateral view of the chest is usually diagnostic.

Figure 8A illustrates another possible diagnostic pitfall. Here the abnormality is perceived as an area of abnormal radiolucency lateral to the right hilum. This finding can be misinterpreted as a cavity, pneumatocele, or other abnormal air collection. In fact, it is caused by pleural fluid tracking into the major fissure and outlining the superior segment of the lower lobe. Because the location and distribution of pleural fluid varies with the patient's position, this "pseudocavity" can appear and disappear over a short period of time on serial portable chest radiographs. The basis of the appearance on the chest radiograph is shown in Figure 8B, a computed tomography (CT) scan of a different patient with a pleural effusion.

This appearance is most commonly observed on a supine or semi-upright portable chest radiograph of patients with moderately large pleural effusions. It is more frequently seen on the right, although it can occur on either side. Diagnostic clues include the location and configuration of the radiolucency, the sharply defined lateral margin with absence of a visible medial margin, and the variable shape of the finding on serial radiographs.[5] The radiolucency can appear, disappear, or change markedly in configuration in a matter of hours. An upright or decubitus view can be performed to exclude a cavitary lung lesion, but it is rarely necessary.

This phenomenon has been attributed to an incomplete major fissure, with the lucent area corresponding to the fused portion of the fissure.[6] However, the margin of the visualized radiolucency actually corresponds to the pleural surface of the superior segment of the lower lobe. Incompleteness of the fissure may play a role by influencing the configuration of the superior segment in the presence of an effusion.

Unusual Air Collections

As with pleural effusions, air collections can assume atypical appearances on portable chest radiographs due to variations in the patient's position and the x-ray beam projection. It is particularly important to be aware that the x-ray beam is rarely completely horizontal in the case of a portable chest radiograph, including those that are labeled as "erect." One important implication of this is that air fluid levels are not likely to be visualized when they exist (Fig. 9). In the case illustrated, the initial semi-erect chest radiograph shows only nonspecific hazing opacity over the left upper lobe, suggestive of consolidation, with some

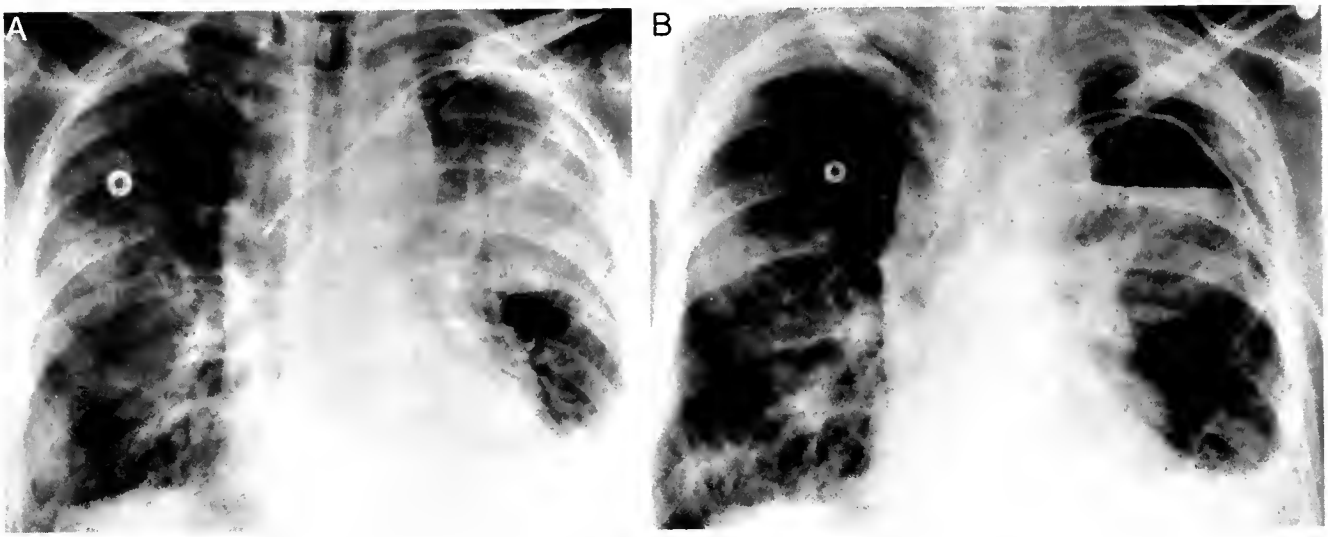

Fig. 9. A: Portable chest radiograph obtained in a semi-erect position shows hazy opacity over the left upper lobe, suggestive of pulmonary consolidation. B: Radiograph obtained later the same day, with a horizontal x-ray beam, clearly shows an air-fluid level in a loculated hydropneumothorax. The air-fluid level was not apparent on the earlier radiograph because the x-ray beam was not horizontally oriented.

poorly defined radiolucency superiorly. A repeat radiograph obtained with a completely horizontal beam clearly shows an air-fluid level within a loculated hydropneumothorax.

For the same reason, free intraperitoneal air cannot be excluded reliably on an "erect" portable chest radiograph. In this situation, it is not the patient's physical position that is critical, but rather the angle of the central x-ray beam.

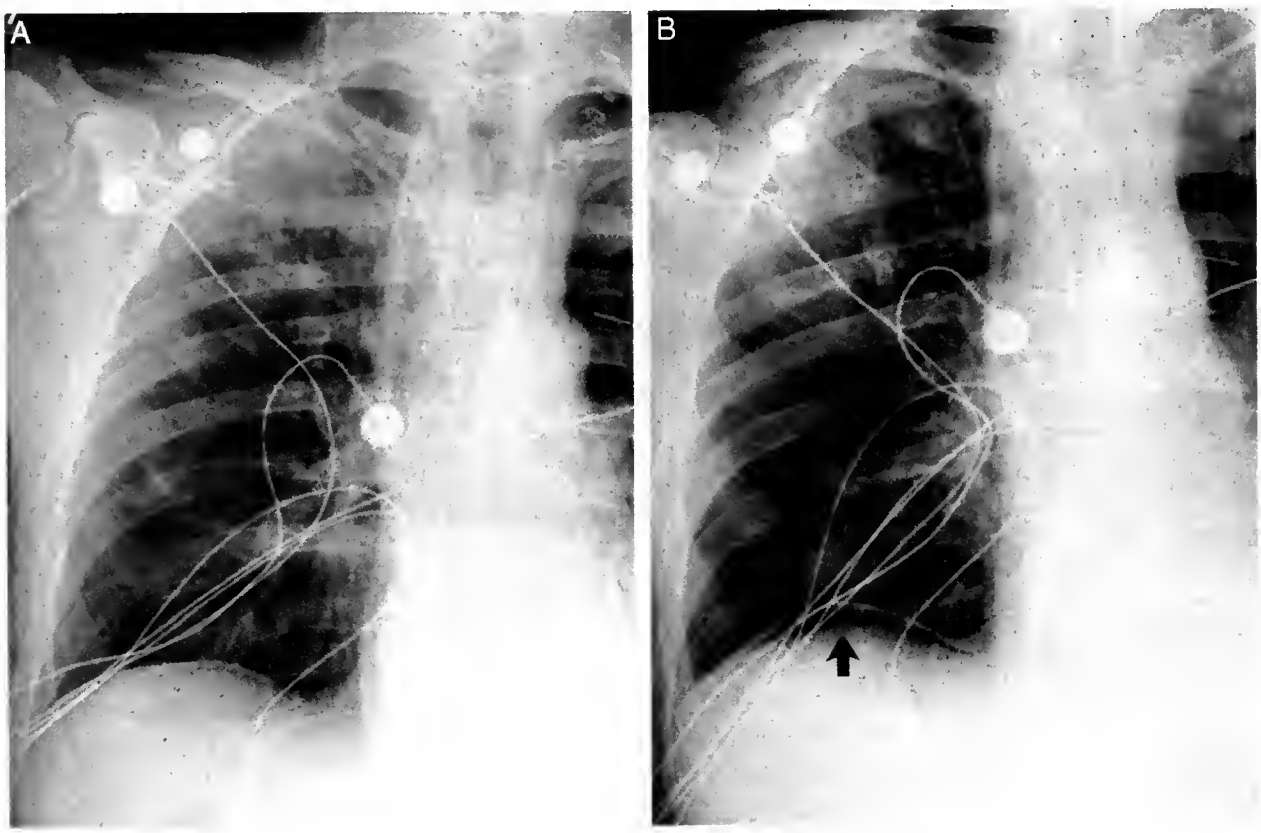

Fig. 10. A: Section of a semi-erect portable chest radiograph showing ill-defined radiolucency beneath the right hemidiaphragm. B: Radiograph obtained with a more horizontal x-ray beam clearly shows free intraperitoneal air (arrow).

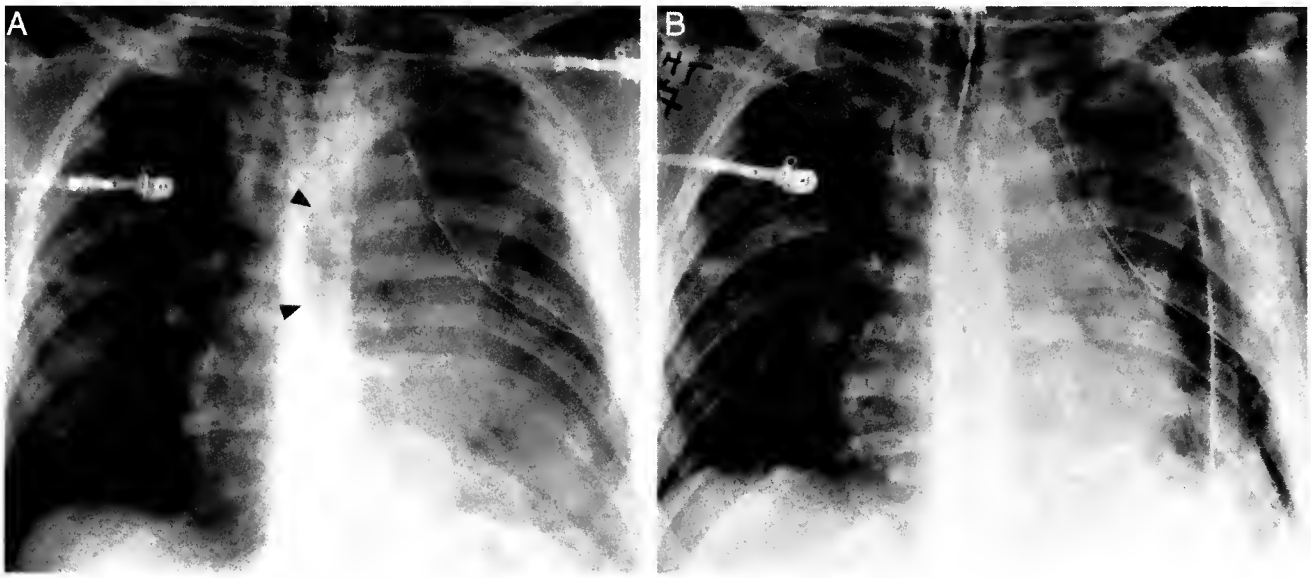

Fig. 11. A: Left anterior loculated pneumothorax. Note evidence of anterior air collection displacing the anterior junction line to the contralateral side (arrowheads). B: Following insertion of a new anterior pleural drain, the pneumothorax has resolved.

Fig. 12. Right subpulmonic pneumothorax. Air is interposed between the base of the lung and the hemidiaphragm.

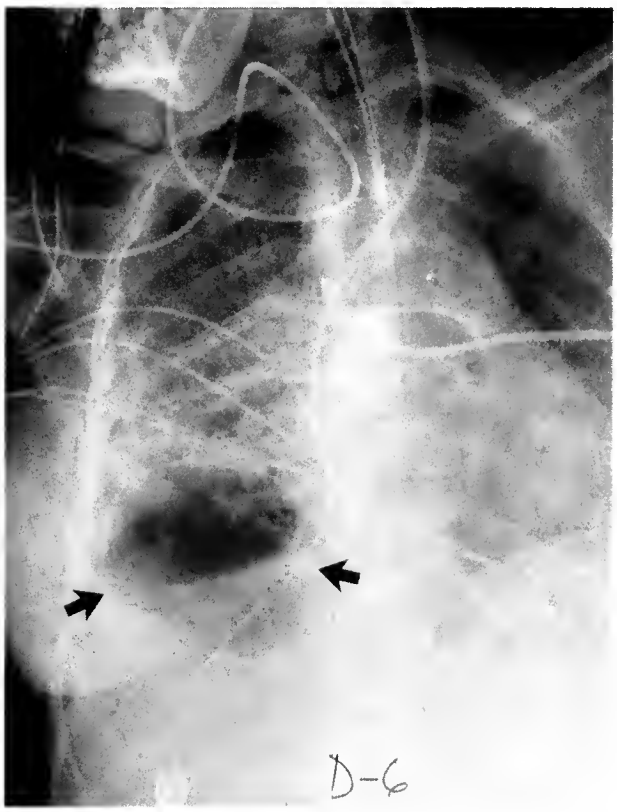

Fig. 13. A rounded radiolucency (arrows) developed overnight in this patient with acute respiratory distress syndrome, receiving assisted ventilation. The appearance and clinical setting are typical of a subpleural air cyst.

Fig. 10 shows a typical semi-erect chest radiograph of a patient with free intraperitoneal air. Only ill-defined lucency is visible beneath the hemidiaphragm. A portable chest radiograph obtained the following day with a more horizontal beam very clearly shows free air. For this reason, when pneumoperitoneum is suspected in a nonambu-

Fig. 14. Chest radiograph showing skin folds (arrows), which can mimic pneumothorax. Several features can be used to distinguish skin folds from pneumothorax (see text).

Fig. 15. Extrapleural air mimicking pneumothorax. Air has dissected from the mediastinum and chest wall, external to the parietal pleura (arrow), in this patient with subcutaneous emphysema.

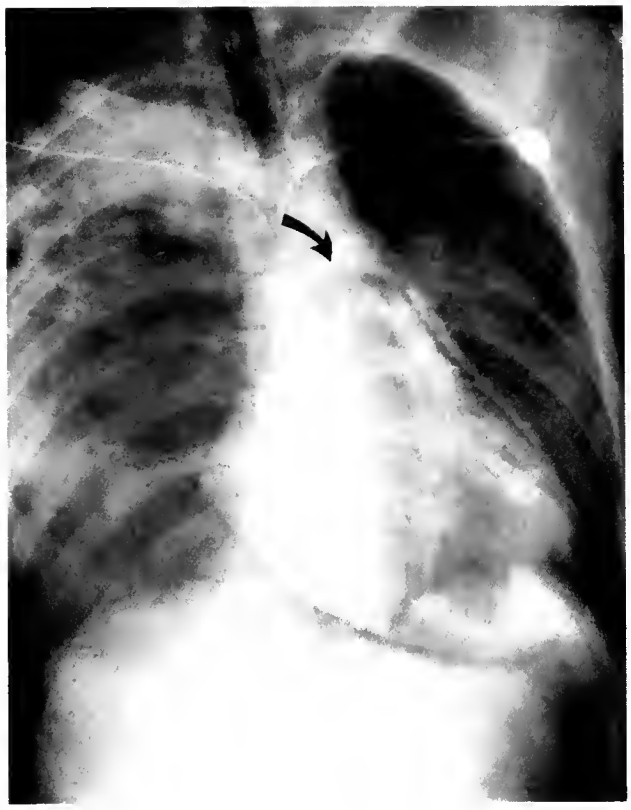

Fig. 16. Pneumopericardium. Postoperative portable chest radiograph following a right pneumonectomy shows characteristic appearance of pneumopericardium. Note limitation of extent of air at superior margin of pericardium (arrow).

Fig. 17. Pneumomediastinum. Air outlines the mediastinum and the central part of the diaphragm (arrowheads).

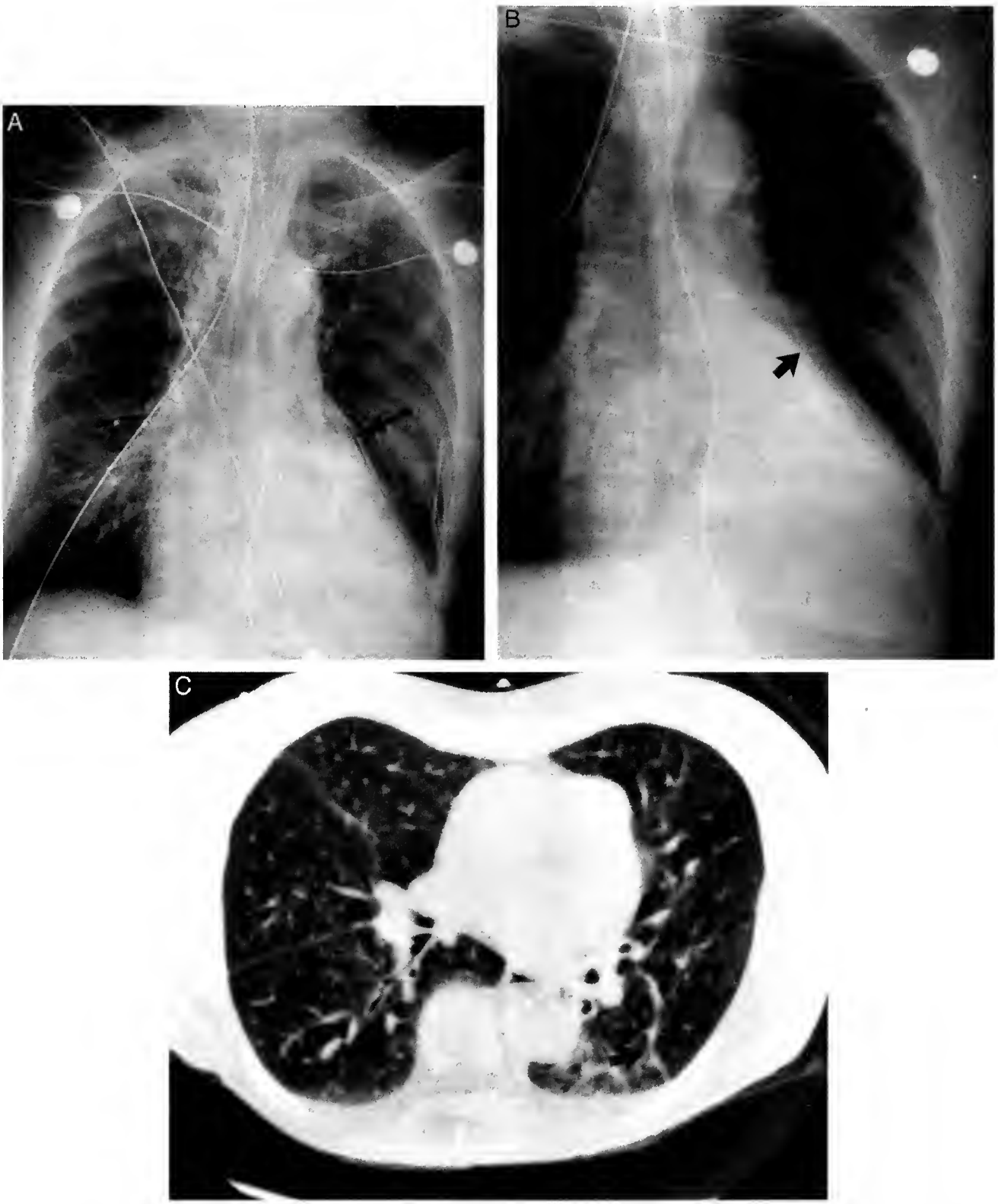

Fig. 18. A: Partial atelectasis of the left lower lobe simulating pneumomediastinum. The major fissure is projected alongside the heart border (arrow). B: A radiograph obtained 2 days earlier shows typical appearance of left lower lobe collapse and consolidation, with posterior and inferior shift of the major fissure (arrow). C: Computed tomography scan shows the major fissure displaced posteriorly (arrow) so that it projects alongside the heart border in a chest radiograph.

Fig. 19. Near total atelectasis of the left lung. Note evidence of volume loss, with mediastinal shift and rotation of the patient toward the ipsilateral side.

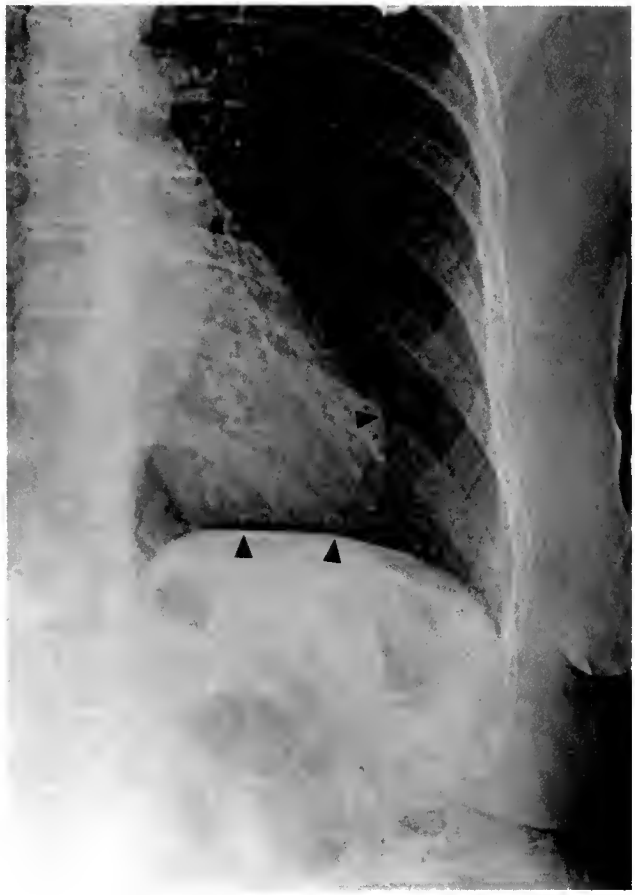

Fig. 20. Overshoot artifact due to edge enhancement. A digital radiograph with unsharp mask (frequency) processing shows an "overshoot" artifact (arrowheads) along the diaphragm and heart border, due to pronounced edge enhancement.

latory patient, a lateral decubitus view of the abdomen should be requested rather than an "erect" portable chest radiograph, because a decubitus view is obtained with a horizontal x-ray beam.

In the case of a typical pneumothorax, the lateral margin and apex of the lung separate from the chest wall. Visualization of the visceral pleura and absence of lung markings peripherally allow for a confident diagnosis. However, in a patient with preexisting pleural adhesions, a pneumothorax will not present in a typical manner. A loculated pneumothorax is often overlooked on portable chest radiographs, because lung markings can extend to the chest wall, and in many such cases a pleural drain is already in place.

A loculated pneumothorax can collect anteromedially, producing a poorly defined area of radiolucency together with evidence of locally increased pressure and volume (mediastinal shift, diaphragmatic depression, and splaying of the ipsilateral ribs). Because these patients often have underlying lung disease and posteriorly layered out effusion, the air collection itself may be obscured and the secondary effects might predominate. In addition, when the air is in a subpulmonic location, a "deep sulcus sign" may be seen because of the lateral costophrenic angle being outlined by the pneumothorax.[7]

Figure 11 illustrates such a case. Note the hazy opacity in the left hemithorax due to posteriorly located pulmonary consolidation and effusion. However, the volume in the left hemithorax is increased and the anterior junction line is displaced toward the right. Following insertion of an anterior pleural drain, the loculated pneumothorax has decompressed. Pneumothorax may loculate in other locations, notably in a subpulmonic position (Fig. 12) or medial to a lower lobe. A subpulmonic pneumothorax must be distinguished from an intra-abdominal air collection or other intrathoracic abnormalities such as subpleural air cysts (Fig. 13). Subpleural air cysts typically occur in patients receiving mechanical ventilation at high pressures. These cysts tend to develop rapidly, over a period of hours, and are prone to rupture into the pleural space, resulting in pneumothorax. A pneumatocele, which is an abnormal cystic space developing in a region of pneumonia, can have a similar appearance. Pneumatocele are quite uncommon in adults, but have been described in 40–60% of cases of pneumonia in children caused by *Staphylococcus aureus*.[8]

Several entities can mimic pneumothorax on a portable chest radiograph. Skin folds can mimic a pneumothorax to

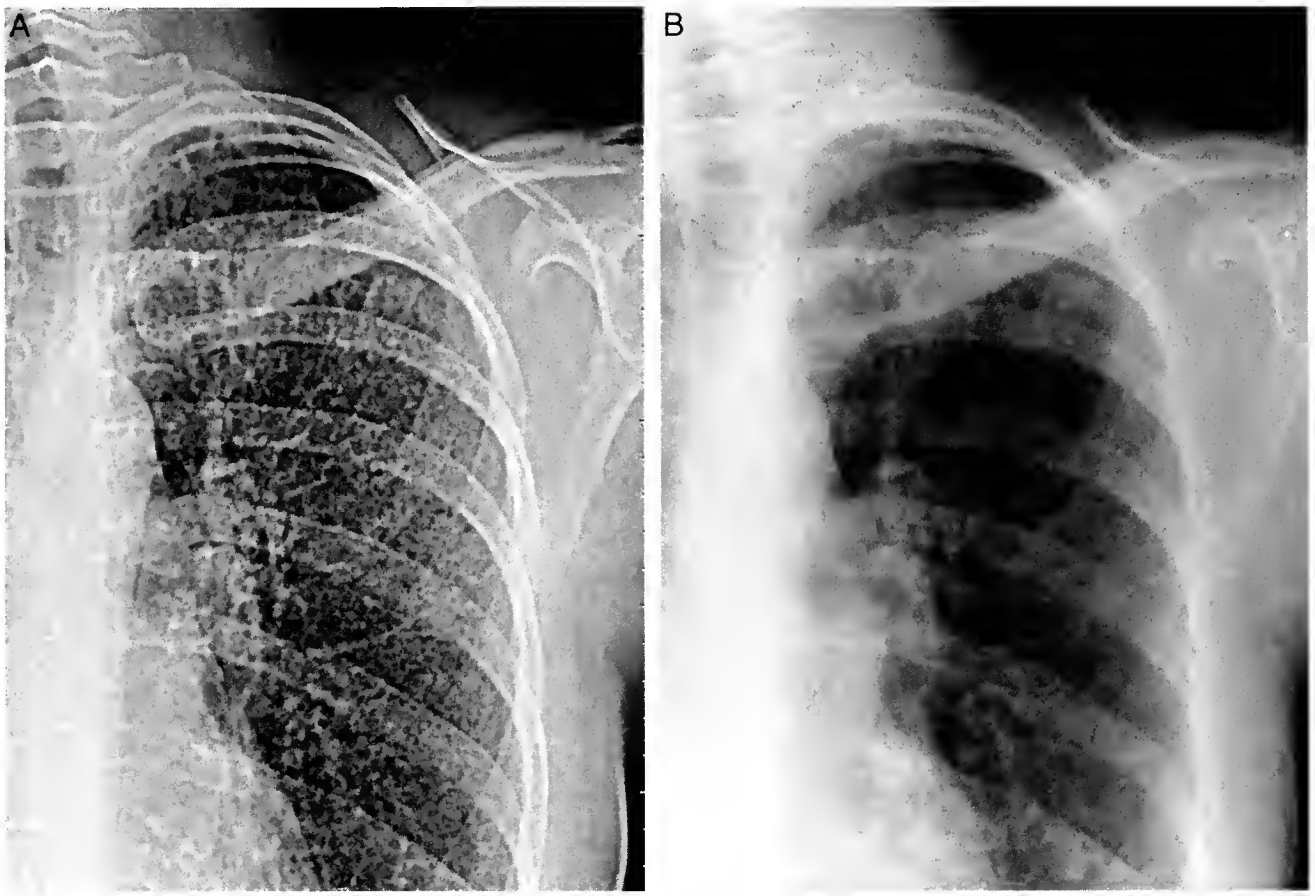

Fig. 21. A: Overenhancement by unsharp mask processing of mild interstitial disease due to sarcoidosis producing exaggerated contrast suggestive of calcifications. B: Reduction of enhancement parameters results in a more easily interpreted image.

a remarkable degree (Fig. 14), but the appearance of a skin fold differs in the following respects.[1] Skin folds rarely follow the entire outline of the suspected lung margin. That is, they either fade out over the lung, or they extend beyond the thoracic margin.[2] Pulmonary markings are often visible peripheral to the skin fold.[3] Skin folds are often multiple and bilateral.[4] The skinfold either appears as a "step" in opacity (lighter medially, darker laterally) or as a dark line, rather than as the fine linear opacity characteristic of the visceral pleura in a true pneumothorax.[5] Skin folds tend to vary from one examination to the next. Therefore a repeat chest radiograph will usually resolve the question.

Recently-removed pleural drains can leave visible tracks that can be mistaken for pneumothorax, though their appearance tends to be characteristic. Comparison with earlier radiographs can usually resolve this question.

In the case of patients with extensive subcutaneous and mediastinal emphysema, air occasionally dissects between the parietal pleura and the chest wall, producing an appearance almost indistinguishable from a small pneumothorax (Fig. 15). Apart from the usual association with subcutaneous emphysema and pneumomediastinum, the lung margin seen in such cases tends to be somewhat irregular and thicker than that in an actual pneumothorax. The location of these air collections is also unaffected by position, which can be more readily seen on a CT scan.

Pneumomediastinum often results from barotrauma in patients receiving mechanical ventilation. The typical appearance is easily recognized, but there are some associated pitfalls. For instance, pneumomediastinum is often misdiagnosed as pneumopericardium. In the ICU setting, pneumopericardium in adults is usually related to recent cardiac surgery or other penetrating trauma. Sudden appearance of pneumopericardium secondary to barotrauma is very unusual in adults, though it is well-documented in neonates.[9] Pneumomediastinum usually presents as abnormal radiolucency at the mediastinal margins and in the soft tissues of the neck. Pneumopericardium, on the other hand, is characterized by its superior limitation by the upper margin of the pericardial sac at the root of the great vessels (Fig. 16). Although a large pneumopericardium can surround the heart and outline its inferior margin, a radiolucent stripe beneath the cardiac silhouette is also common

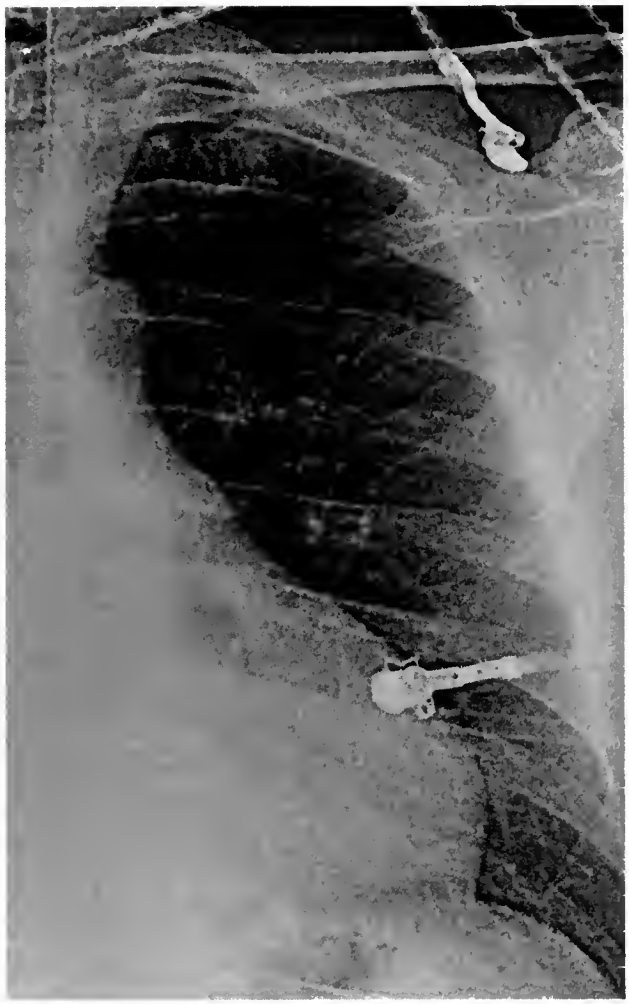

Fig. 22. Quantum mottle due to underexposure of a digital radiograph. This radiograph was accidentally underexposed with a dose equivalent to approximately 20% of the normal dose for a 250 speed screen/film system. Although large structures are still visible, subtle details are degraded by severe mottle, which produces a grainy appearance.

in pneumomediastinum due to air dissecting beneath the pericardium ("continuous diaphragm sign") (Fig. 17).

Atelectasis

Lobar and subsegmental atelectasis are among the more common causes of pulmonary opacity in ICU patients. Although distinction between atelectasis and other causes of consolidation cannot always be made, atelectasis of a lobe or of an entire lung can be confidently identified in most cases. Still, failure to recognize major atelectasis is a common error.

Any phenomenon that causes a radiolucent halo alongside the mediastinum can be mistaken for pneumomediastinum. One such example would be a small medially loculated pneumothorax.

One particular pitfall involves partial lower lobe atelectasis. When a lower lobe collapses, the major fissure rotates posteriorly, until it becomes oriented so that it is sharply defined in the AP projection.[10] In such cases, partial aeration of the collapsed lobe can result in projection of the major fissure lateral to and parallel with the heart border. This phenomenon is more commonly seen in the left side and, in the appropriate setting, can simulate pneumomediastinum or pneumopericardium (Fig. 18). The key to the correct diagnosis lies in recognition of the underlying lobar atelectasis manifested by retrocardiac opacity and volume loss. As is often the case in portable chest radiography, comparison with previous sequential radiographs can be very helpful.

When an entire hemithorax becomes opaque overnight, the differential lies between atelectasis and pleural effusion. In practice there is often a combination of both, though one or the other is the dominant problem. The distinction can be made based on the volume of the affected hemithorax: if volume is decreased with mediastinal shift, diaphragmatic elevation, and rib approximation, the problem is probably related to bronchial obstruction by a mucous plug (Fig. 19). Absence of air bronchograms support this diagnosis. On the other hand, if the ipsilateral volume is increased, this favors a pleural effusion with secondary compressive atelectasis of the underlying lung. In such cases, central air bronchograms are often visible.

Lobar atelectasis can be misinterpreted as pneumonia or other nonspecific consolidation if the associated volume loss is not recognized. This error is particularly common in portable radiography in the case of left lower lobe collapse. The atelectatic lobe creates a retrocardiac opacity with associated mediastinal shift. Invariably, the patient's thorax rotates toward the side of the collapse, probably secondary to elastic recoil of the chest wall. In such cases, the mediastinal shift tends to be attributed to rotation, and the other signs of atelectasis might be overlooked.

Digital Radiography

Digital radiography using photostimulable phosphor plates is increasingly used for portable chest radiography, because the wide exposure latitude of these systems results in more consistent image quality and reduced repeat rates. The ability to store the images digitally for review on workstation monitors in the ICU or emergency room is an added benefit.[3] However, digital radiography also can produce some unique artifacts. Some of these are produced by digital unsharp mask processing. Unsharp mask processing, also known as frequency processing, is a technique that increases the contrast of small structures in a designated frequency range within the image. It also produces an edge enhancement effect that accentuates and sharpens boundaries between structures of differing attenuation. This

can produce an appearance of abnormal radiolucency at the mediastinal and diaphragmatic margins of the lung, which can be mistaken for a pneumomediastinum or subpulmonic pneumothorax (Fig. 20). On the other side of these boundaries, an opaque stripe can sometimes be seen, simulating diaphragmatic or mediastinal calcified pleural plaques. In the lungs, the most common pitfall associated with digital image processing is a tendency for inexperienced observers to overdiagnose interstitial lung disease because of the accentuation of normal lung markings. In the case of fine nodular interstitial disease, excessive frequency processing can produce an appearance of "pseudocalcification" because of high local contrast[11] (Fig. 21). These effects can be modified by appropriate selection of processing parameters and by familiarity with their effects.

Another artifact that may be confusing for the inexperienced reader can arise from underexposure of a digital radiograph. Because digital systems are designed to compensate for underexposure and overexposure, a digital radiograph performed at a very low dose will not appear light or "underexposed." Rather, the signal will be amplified to achieve a standard level of opacity. The effect on the image will be similar to that of using a very high speed photographic film. That is, it will appear grainy or mottled because of the relatively small numbers of x-ray photons detected by the phosphor plate (Fig. 22). In imaging parlance this is referred to as a "noisy" image, and noise due to underexposure is called "quantum mottle." This phenomenon limits the degree to which radiation dose can be reduced with digital systems.

Summary

Certain measures can be effective in reducing the number of potential pitfalls in the interpretation of bedside x-ray examinations. These include standardization of technique, awareness of relevant technical issues, interpretation in the context of multiple prior images, and familiarity with the effects of projection and position on the appearances of cardiopulmonary abnormalities.

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Chest Imaging in the Cardiac Intensive Care Unit

Ella A Kazerooni MD and Philip N Cascade MD

Introduction
Pulmonary Parenchymal Opacification
Evaluation of Cardiovascular Status
Cardiogenic Edema versus Other Causes of Lung Opacity
Vascular Catheters and Devices
Commonly Used Devices
Advanced Cardiovascular Support Devices

[Respir Care 1999;44(9):1033–1043] *Key words: chest radiography, radiograph, computed tomography, heart disease, catherization, extracorporeal membrane oxygenation, respiratory distress syndrome.*

Introduction

Chest imaging in the cardiac intensive care unit (CICU) plays an important role in the successful management of critically ill patients, whether studies are obtained as "routine" each morning or as "urgent/stat" examinations because of abrupt changes in clinical status or manipulation of vascular catheters, pacemaker leads, or monitoring devices. Several studies have found that obtaining daily bedside chest radiographs of intensive care unit (ICU) patients detects unexpected problems and alters the treatment or diagnostic approach after 37–65% of routine chest radiographs.[1–3] Essential elements of ICU radiology include timely performance and accurate interpretation of examinations; prompt communication of abnormalities to the requesting clinicians; and teamwork between radiologists, radiology technologists, intensive care clinicians, nurses, and respiratory therapists. On-line radiograph interpretation and daily radiology rounds between the CICU and radiology services can add to the quality of care.

Comprehensive assessment of chest radiographs in the CICU includes evaluation of visible cardiovascular structures and physiologic parameters, lung parenchymal opacification, pleural disease, and confirmation that indwelling catheters, tubes, and monitoring devices are positioned correctly. Musculoskeletal structures and the visualized portion of the abdomen should also be assessed. The interpretation of CICU radiographs is made more difficult by the limited power output of portable equipment and resulting inconsistent radiographic technique. Patient factors such as inability to obtain motion-free radiographs at full inspiration, inability to position patients upright, and overlying devices further impair image quality. The present article addresses topics pertinent to bedside radiology in the CICU.

Pulmonary Parenchymal Opacification

Evaluation of Cardiovascular Status

Chest radiographs are useful when assessing cardiovascular status. Systemic blood volume, pulmonary blood volume, central venous pressure, pulmonary arterial pressure, capillary wedge pressure, and extravascular water should be evaluated, and the findings correlated with clinical symptoms and physical findings.

Systemic Blood Volume. Systemic blood volume is assessed by examining the "vascular pedicle" of the superior mediastinum.[4] The right margin of the pedicle is the lateral aspect of the superior vena cava (SVC); the left margin is the lateral aspect of the left subclavian artery as it arises from the aortic arch. These distensible vessels respond to changes in circulating blood volume. Serial portable radiographs obtained with consistent positioning may show changes in pedicle width, an important fluid status indi-

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cator. The transverse cardiac diameter also responds to changes in circulating blood volume, but is less predictable. A 66% increase in volume is required to produce unequivocal radiographic change in left ventricular chamber size.[5]

Central Venous Pressure. The azygous vein responds directly to right atrial pressure because of its distensibility, and responds less directly to changes in circulating blood volume. This can be assessed by changes in the relative width of the azygous vein, seen end-on just above the right main bronchus.

Pulmonary Blood Flow Volume. Pulmonary blood flow volume (PBF) is estimated by examining the relative diameter of pulmonary vessels, and can be frustrating on portable chest radiographs; but with careful study blood flow patterns can be recognized, and interpretation improves with experience. The ratio of the diameter of adjacent bronchi and pulmonary arteries seen end-on aids in determining whether vessels are enlarged.[6] Under normal circumstances, the pulmonary artery to bronchus ratio (A:B ratio) is 1:1 or less. The A:B ratio is particularly well seen as the end-on anterior segmental artery and bronchus of each upper lobe. If the A:B ratio is increased throughout the lungs, PBF is increased; a generalized decrease indicates reduced PBF.[7] Consider 3 examples:

1. Fluid overload: incremental widening of the vascular pedicle with increased PBF and increased transverse cardiac diameter.
2. Cardiac tamponade: decreased PBF and a wide vascular pedicle suggest cardiac tamponade. The reduced right ventricular output reduces PBF, and the wide vascular pedicle is due to obstruction of central venous return.
3. Left-to-right intracardiac shunt: increased PBF with normal or decreased systemic blood volume suggests a left-to-right shunt such as a septal defect or acquired rupture of the ventricular septum following myocardial infarction.

Pulmonary Arterial Pressure. Pulmonary arterial pressure can be estimated by comparing the diameter of central and peripheral pulmonary arteries. Depending on the method used, correlation coefficients of up to 0.87 have been reported.[6] These observations do not distinguish acute variations in pulmonary artery pressure in unstable patients, but predict a range of pressures in the steady state due to chronic increased vascular resistance.

Left Ventricular Function. Left ventricular function is estimated by analyzing pulmonary vessels and the lung interstitium. In upright patients with normal cardiovascular function, the diameter of the lower lobe vessels is larger than in the upper lobes, with preferential blood flow to the lung bases because of the effect of gravity. Vessel margins are normally sharply demarcated. In this state, total extravascular lung water is approximately 50 mL/L at total lung capacity.[7,8] Several studies have observed changes in blood vessel size and distinctness with elevations of pulmonary capillary wedge pressure (PCWP).[9,10] Ideally, this can be divided into 3 stages: cephalization, interstitial edema, and alveolar edema.

As PCWP rises above normal (mean 12 mm Hg), vessel diameters in upper and lower lung zones equalize. With further increase in PCWP, pulmonary blood flow redistributes to the upper lung zones (cephalization), recognized as larger vessels in the upper lungs compared to lung bases.

When PCWP rises to 18–25 mm Hg, the colloid osmotic pressure of blood is exceeded and fluid transudates into the lung interstitium. Interstitial fluid around blood vessels results in blurred vessel margins or perivascular indistinctness. Similarly, interstitial fluid around bronchi results in peribronchial cuffing, and fluid in the interlobular septa creates linear opacities that extend to the pleural surface (Kerley B lines). Kerley A lines are longer and central, and Kerley C lines are finer and at the lung bases (rarely recognized on portable radiographs). At total lung capacity, total lung water is approximately 60–100 mL/L.[9,10]

With a PCWP of $\geq$ 25 mm Hg, transudates collect in large amounts in the interstitium and alveoli, producing alveolar pulmonary edema. This is recognized as fluffy, symmetric lung opacity, particularly perihilar and basilar. When alveolar fluid begins to appear, lung water is estimated to be 110–130 mL/L; with widespread edema this rises to 160 mL/L or more.[9,10]

Frequently there is a discrepancy between PCWP measurements and the degree of heart failure evident on chest radiographs. Herman et al found that 38% of patients with left ventricular end-diastolic pressures $>$ 20 mm Hg had no radiographic evidence of left heart failure.[11] Others have noted discrepancies in the estimation of PCWP in subgroups of patients with acute myocardial infarction and acute left ventricular power failure.[11,12] Possible explanations for this include preexisting emphysema, interstitial lung disease, or poor inspiration at the time of the radiograph. Furthermore, hemodynamic measurements reflect a moment in time, while movement of water in and out of the extravascular compartment may take hours to days; therefore the resorption of the fluid from the lung and lung opacity on radiographs may lag behind normalization of PCWP.

Many of the above estimations were made on nonportable radiographs. Interpreting portable radiographs is less exact. It is not possible to estimate blood flow in the upper versus the lower lungs, because critically ill patients are usually not examined fully upright. Woodring attempted to circumvent this problem by studying the relationship of

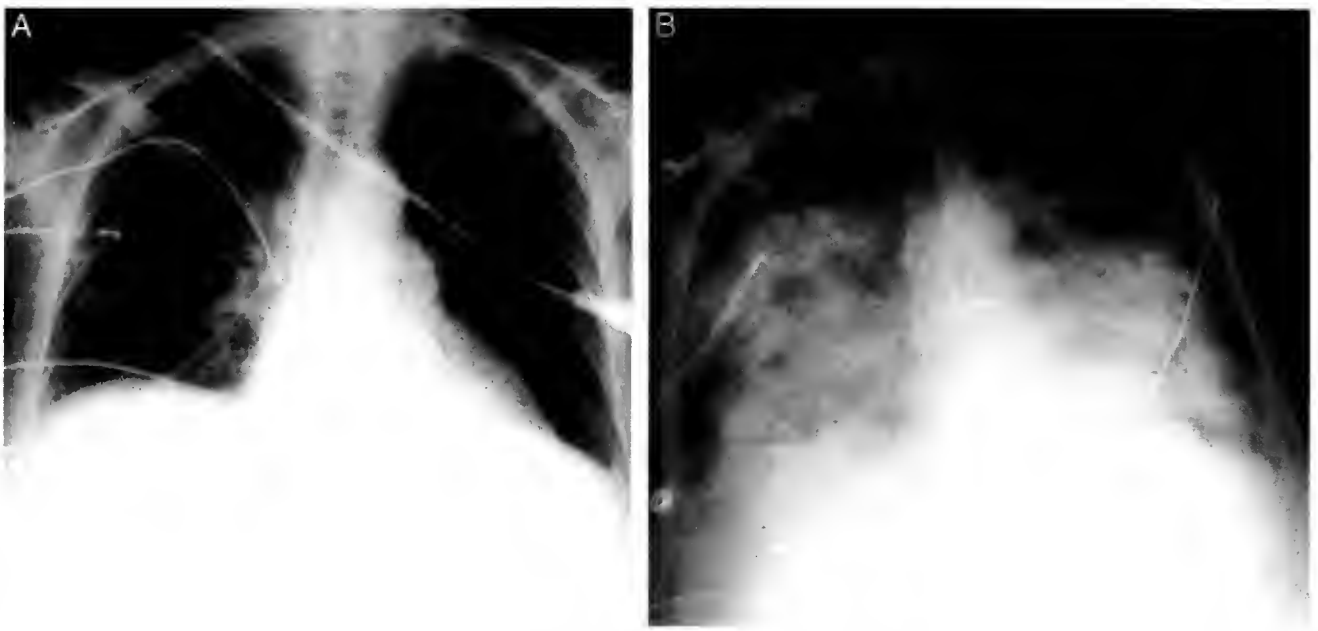

Fig. 1. Acute cardiogenic pulmonary edema in a 68-year-old woman presenting to the emergency room with chest pain. A: Initial chest radiograph shows normal heart size and normal lungs. B: Chest radiograph 2½ days later shows acute perihilar or "butterfly" and lower lobe ill-defined alveolar opacity, consistent with acute edema.

pulmonary artery diameter to the adjacent bronchus in supine and upright patients.[13] In a supine patient, hydrostatic differences occur from the dorsal to the ventral aspect of the lungs rather than from the lung bases to the lung apices. Under normal conditions, the A:B ratio is equalized throughout the lungs. When PCWP is elevated, the ratio increases throughout the lungs, allowing distinction between left ventricular failure and normal physiology. This ratio cannot distinguish increased PBF (cardiac shunt) from left ventricular failure, as both produce an increase in the A:B ratio. However, in practice this comparison is often limited by the inability to identify adjacent end-on arteries and bronchi.

Extravascular Soft Tissue Water. Extravascular soft tissue water is estimated by changes in chest wall thickness and can only be used when patients are reproducibly positioned. A retrospective study correlating changes in chest wall thickness with body weight in renal failure patients found a correlation coefficient of $r = 0.56$.[7]

Cardiogenic Edema versus Other Causes of Lung Opacity

Common etiologies of lung parenchymal opacity in the CICU other than cardiogenic pulmonary edema include atelectasis, pneumonia, and, less commonly, acute respiratory distress syndrome (ARDS). On a single radiograph it may be difficult to distinguish between these, particularly if the abnormality is diffuse bilateral alveolar abnormality. Pulmonary edema may be predominantly perihilar, often described as "batwing" or "butterfly" in appearance (Fig. 1), which is not typical of ARDS or pneumonia; however, the lack of this feature is not helpful. On serial radiographs, cardiogenic edema usually gets better or worse over hours to a day or so, whereas the etiology of left ventricular failure is clinically identified (see Fig. 1). If left ventricular failure is treatable with pharmacologic interventions or coronary artery revascularization, it clears on radiographs. If the left ventricle is severely and irreversibly damaged (massive acute myocardial infarction, severe progressive cardiomyopathy), there is progressive edema and probable death, unless an assist device can provide support until the left ventricle recovers or heart transplantation occurs.

Some patients may have atypical radiographic manifestations of pulmonary edema. Pulmonary edema changes with position. A patient in the Trendelenburg position will have more severe edema in the upper lobes (Fig. 2A), compared to a patient who is sitting upright (Fig. 2B). A patient who preferentially lies on the left side will develop more severe edema in the dependent left lung. Also, in patients with severe upper lobe-predominant emphysema at baseline, most of the pulmonary artery blood flow goes to the lower lobes, where gas exchange can occur with more normal lung. When left ventricular failure occurs, edema manifests as bibasilar alveolar consolidation, mimicking pneumonia or aspiration (Fig. 3). Similarly, patients

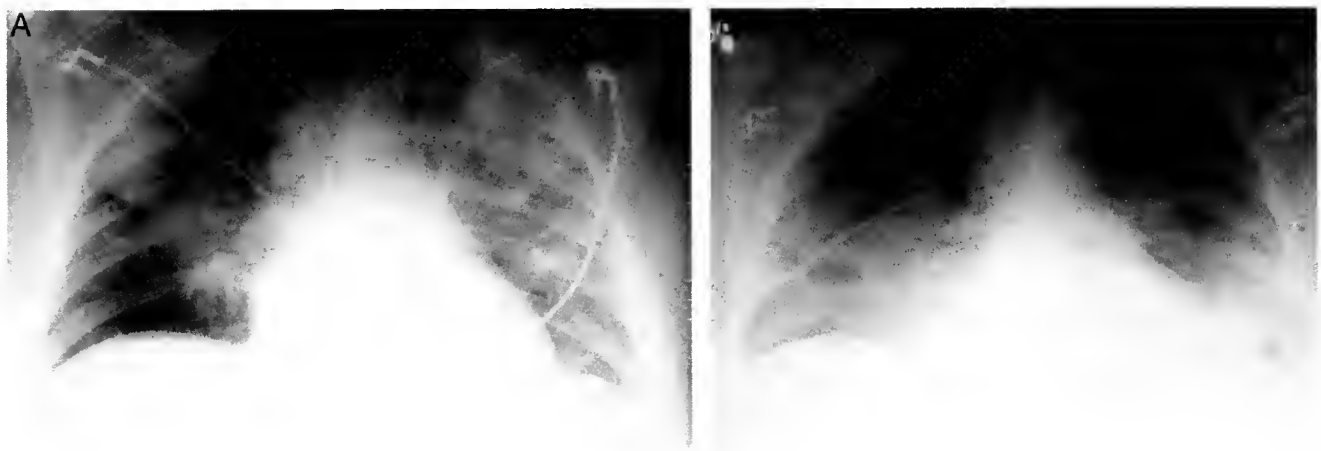

Fig. 2. Positional pulmonary edema in a 35-year-old man secondary to overhydration. A: Chest radiograph taken in the Trendelenburg position shows upper-lobe-predominant edema. B: Chest radiograph taken 19 hours later, after resting predominantly in a semiupright position, shows perihilar and lower-lung-predominant edema.

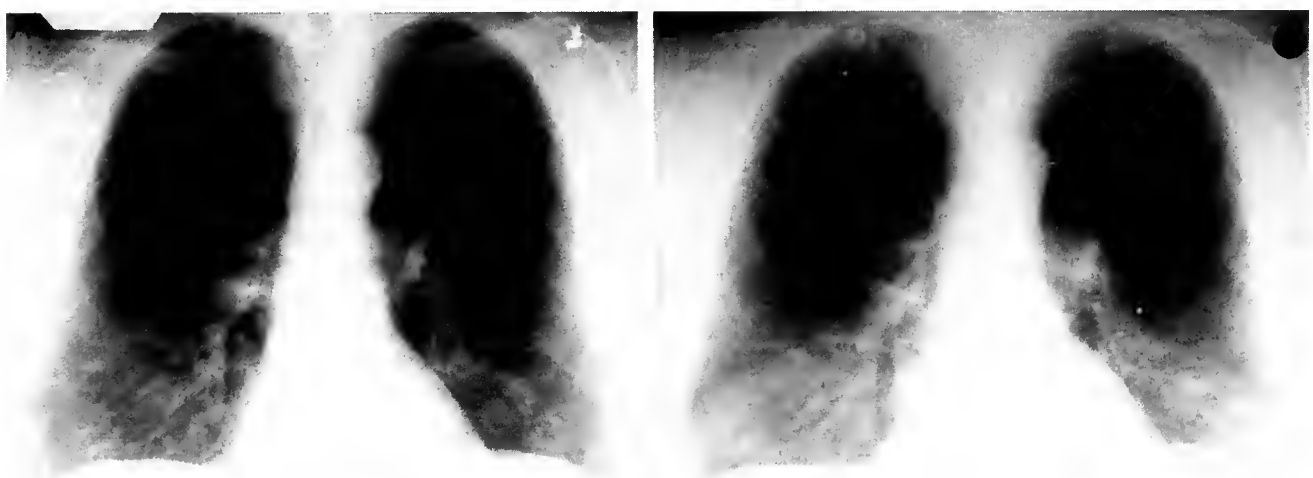

Fig. 3. Atypical pulmonary edema in a 73-year-old man with upper-lobe-predominant emphysema, with shunting of pulmonary blood flow at rest from the lung apices to the bases in order match perfusion with the greater ventilation in the lower lungs. A: Baseline chest radiograph shows hyperinflated and hyperlucent lungs, consistent with emphysema. B: Chest radiograph when the patient is in left ventricular failure shows lower-lobe-predominant alveolar opacity, which mimics pneumonia or aspiration.

with pulmonary embolism or a tumor occluding a pulmonary artery branch will not develop edema in the lung parenchyma supplied by the occluded vessel(s) (Fig. 4).

Atelectasis. In contrast to edema, diffuse atelectasis can often be recognized by secondary signs of volume loss, including low lung volumes and crowding of bronchovascular structures and the ribs. More commonly, focal basilar subsegmental atelectasis occurs, often transiently. The left lower lobe is the most common location for both atelectasis and pneumonia following cardiac surgery, secondary to stretching and cold-induced injury of the phrenic nerve.[14] Radiographic technique is important, as underpenetrated radiographs result in overcalling abnormality. With as little as 10 degrees lordotic angulation of the x-ray beam, the beam is no longer tangential to the apex of the hemidiaphragm, and pseudoconsolidation may appear behind the heart.[15]

Infection. The radiographic hallmark of pneumonia is airspace consolidation with air-bronchograms, which may be segmental, lobar, or diffuse. Pneumonia is difficult to diagnose in the critically ill, because fever and leukocytosis, the hallmarks of infection may be absent. Fever is not a reliable indicator of infection and is present in at least 50% of patients with atelectasis and no pneumonia.[16] The radiographic false negative misdiagnosis rate is espe-

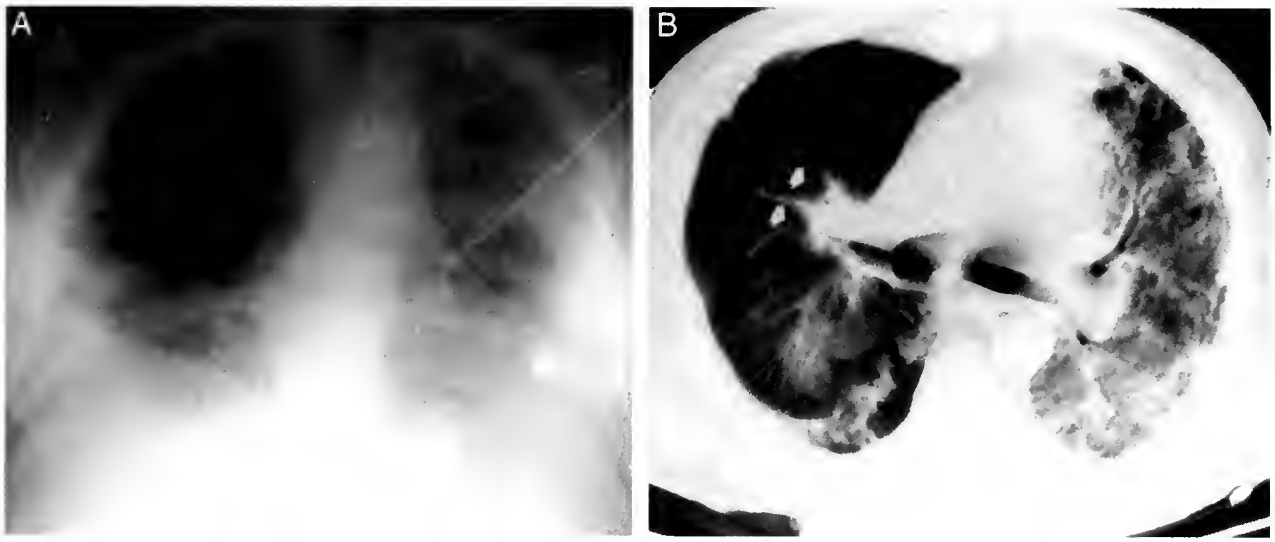

Fig. 4. Atypical pulmonary edema secondary to occlusion of the right upper lobe pulmonary artery by a central lung cancer in a 50-year-old man. A: Chest radiograph shows diffuse bilateral alveolar opacity of edema, sparing the upper portion of the right lung. B: Computed tomography image at the level of the carina demonstrates diffuse alveolar ground glass attenuation of edema sparing the right upper lobe, with a central spiculated lung cancer (arrows) located centrally near the right upper lobe pulmonary artery.

cially prevalent in the setting of ARDS, where it may be as high as 29%.[17] Mock et al reviewed the clinical variables and radiographic findings of 80 patients with positive sputum cultures.[18] The chest radiograph was scored with 1 point each for the presence of new airspace shadows, air-bronchograms, segmental infiltrates, asymmetric infiltrates, infiltrates in nondependent lung, ipsilateral pleural effusion, and for the absence of volume loss, cardiomegaly, and hilar enlargement. Patients with radiographic scores of 4–10 were more likely to have positive blood and fluid cultures, polymicrobial cultures, multisystem organ failure, *Escherichia coli* or pseudomonas infection, and improvement on antibiotics, than patients with low score radiographs. Clinical symptoms of fever, leukocytosis, respiratory failure, and mortality did not correlate with the radiographic findings.

Patchy bilateral parenchymal opacities (which typically progress slowly but can occasionally progress rapidly) can mimic pulmonary edema, but should raise the possibility of septic pulmonary emboli. The source of infection can arise from cardiac valvular vegetations, an indwelling catheter, the urinary tract, a wound, or an abscess.[19] Computed tomography (CT) is more sensitive than plain radiography in the detection and diagnosis of septic emboli, and may yield a diagnosis before it is clinically or radiographically suspected.[20,21] On plain film the classic findings of septic pulmonary emboli include peripheral wedge-shaped opacities and nodules with or without cavitation, whereas the chest radiograph can be normal or show nonspecific opacities.[21] On CT, the multiple pulmonary nodules range in size from 0.5–3.5 cm, 50% associated with cavitation; 39% of patients also had pleural effusions.[21]

Acute Respiratory Distress Syndrome. While the diagnosis of ARDS is based primarily on clinical findings, the chest radiograph may provide additional diagnostic information concerning the effectiveness of treatment and prognosis. In 1977 Bachofen and Weibel described the 3 pathologic phases of ARDS lung injury and response.[22] The acute phase—represented pathologically by diffuse endothelial cell injury with alveolar capillary leak of proteinaceous fluid and neutrophils—manifests as diffuse ill-defined alveolar opacities, predominantly in the periphery of the lungs.[23] As capillary leak progresses with greater fluid extravasation from the intravascular space into the alveoli, widespread pulmonary opacification and, in certain cases, complete "white-out" of the lungs occur.[24] With injury to alveolar epithelial cells, there is decreased surfactant production and decreased lung compliance, reflected on radiographs as reduced lung volume and atelectasis.[22]

In general, the alveolar opacities of ARDS progress over several days and subsequently change very slowly from day to day. In contrast to cardiogenic, uremic, and hypervolemic pulmonary edema, the alveolar edema of ARDS is not associated with widening of the vascular pedicle, cardiomegaly, or altered pulmonary blood flow distribution. When the pulmonary vessels can be distinguished, they are often constricted. Since capillary leak occurs directly into the alveolar spaces, septal lines are usually absent. In the subacute phase (occurring over the

next 5–10 days) the pathologic process of proliferation of epithelial cells and fibroblasts, as well as collagen deposition, produces the radiographic findings of progressive lung destruction and a transition from alveolar to interstitial opacities. Evidence of barotrauma, including pneumothorax and pneumatocele, is frequently found.[25,26] While some patients recover from ARDS with no pulmonary function deficit, other patients eventually enter the chronic phase several weeks after the initial lung injury, manifested on chest radiographs by fibrosis and focal emphysema.

Vascular Catheters and Devices

More often than not, catheters, tubes, and indwelling monitoring devices used in CICU patients are initially placed in the correct position, but the interpreting radiologist and clinician should bear in mind the possibility of initial misplacement or subsequent movement of these devices. Most catheter misplacements are readily correctable and without consequence; others are serious and require immediate intervention. Bekemeyer reported that 27% of lines and tubes were malpositioned on portable radiographs taken after initial placement, and that in 20% the radiograph was instrumental in deciding to reposition the catheter.[1] Temporary circulatory assist devices are commonly used in the CICU, and permanent devices may soon be available. Knowledge of the radiographic appearance of these devices is necessary to evaluate for correct position and to identify complications.

The demand for donor hearts in 1987 was approximately 15,000, with a potential donor pool of 400–1,100.[27] In 1993 the need was estimated to be > 40,000 hearts each year, with a potential donor pool of approximately 5,200.[28] By the year 2010 there may be 35,000–70,000 candidates for heart transplantation or permanent device implantation.[29]

Commonly Used Devices

Central Venous Catheters. Common complications of central venous catheter (CVC) placement include pneumothorax and catheter malposition. Catheter tips can be placed blindly into almost any reachable vein, including up the jugular vein into the neck, across the mediastinum, down the axilla, azygous vein, internal mammary vein, left-sided SVC, or the inferior vena cava. Knowledge of normal venous anatomy and common variants is important in localizing these catheters. Portable chest radiographs are obtained after CVC placement, both to confirm that the catheter tip is in good position in a brachiocephalic vein or the SVC, and to rule out pneumothorax (Fig. 5). Less common complications are venous perforation with mediastinal and/or neck hematoma, inadvertent arterial puncture or arterial placement (Fig. 6), and fracture of the CVC with retained fragment. Arrhythmias can occur if the catheter tip resides in the right atrium. While thrombosis and infection are common complications, there is usually no evidence on chest radiographs. Dilatation of the azygous vein may indicate a development of a collateral pathway if there is SVC thrombosis.[30] Scott et al reviewed the reasons for 170 injuries (n = 118) or deaths (n = 52) secondary to central venous catheter placement reported to the federal Food and Drug Administration Medical Device Reporting

Fig. 5. Normal position of a central venous catheter placed via the left subclavian vein (arrowheads) with tip in the superior vena cava.

Fig. 6. Arterial misplacement of a right subclavian central venous catheter, passing through the aortic arch with tip (arrow) in the proximal descending aorta.

Fig. 7. Pulmonary artery pseudoaneurysm secondary to a Swan-Ganz catheter that had been positioned too distally into a branch of the right pulmonary artery. A: Intravenous contrast-enhanced chest computed tomography image at the level of the right pulmonary artery demonstrates a 1.5 cm round structure (arrowheads) immediately adjacent to and enhancing as much as the right pulmonary artery, consistent with a pseudoaneurysm. B: Pulmonary angiogram similarly demonstrates the rounded 1.5 cm structure to fill with contrast. C: Pulmonary angiogram after embolization demonstrates complete occlusion of the pseudoaneurysm.

Program (1984–1986).[31] Fifty-one percent were related to health care professionals (technique), 12% were due to device failure, 3% were patient-related, 3% were pathologic/physiologic (thrombosis or thromboembolism), and 28% were for unknown reasons. Vascular perforation was responsible for 94% of fatalities and 54% of injuries. The

Fig. 8. Normal position of a Swan-Ganz catheter (arrows) entering via the right subclavian vein with tip in the right pulmonary artery.

most common reasons cited for catheter fracture were excessive force during catheter placement or removal; excess syringe pressure in an attempt to open an occlusion; shearing the catheter with the insertion needle; puncturing the catheter with a surgical needle; and failure to follow insert directions.

Swan-Ganz Catheters. In addition to the risk of pneumothorax and bleeding associated with central venous catheter placement, there are several complications unique to flow-directed Swan-Ganz catheters, including vessel perforation with pulmonary hemorrhage, pulmonary infarcts (uncommon due to the dual arterial supply of the lung from bronchial and pulmonary arteries), and pulmonary artery pseudoaneurysms (Fig. 7).[32] Ideally, the catheter tip should reside within a large pulmonary artery branch, not peripheral to the interlobar pulmonary artery that forms the comma-shaped structure of each hila (Fig. 8). The balloon should not be inflated except during placement and pressure measurement. Given the length of catheter within the cardiovascular system, it is not uncommon for catheters to become coiled in the heart (Fig. 9), or sometimes tied in a knot (Fig. 10).

Intra-aortic Balloon Pump. The intra-aortic balloon pump (IABP) is primarily used to improve cardiac function in the case of left ventricular failure, particularly for cardiogenic shock, unstable angina, and myocardial infarction with or without septal rupture. The IABP, placed through the common femoral artery under fluoroscopic guidance, supplements cardiac output by 20–30%. The balloon inflates in diastole, increasing pressure in the proximal aorta and increasing coronary perfusion. Occasionally, the radiolucency of the inflated balloon can be identified on chest radiographs (Fig. 11). The balloon deflates in systole to reduce left ventricular afterload and myocar-

Fig. 9. Swan Ganz catheter coiled with multiple loops of redundant catheter in the right atrium and right ventricle (arrows).

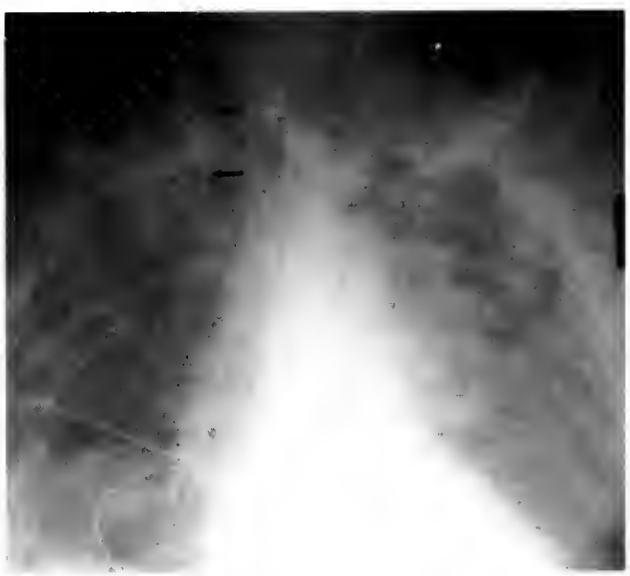

Fig. 10. Swan-Ganz catheter placed via a right subclavian approach passes up the internal jugular vein (arrows) rather than centrally into the brachiocephalic vein, and is coiled in a knot at the tip (arrowhead).

Fig. 11. Normal position of an intra-aortic balloon pump. Note the radiopaque tip (arrowhead) positioned at the inferior aspect of the aortic knob, and the less commonly seen vertical lucency (arrows) of the inflated balloon in the descending thoracic aorta.

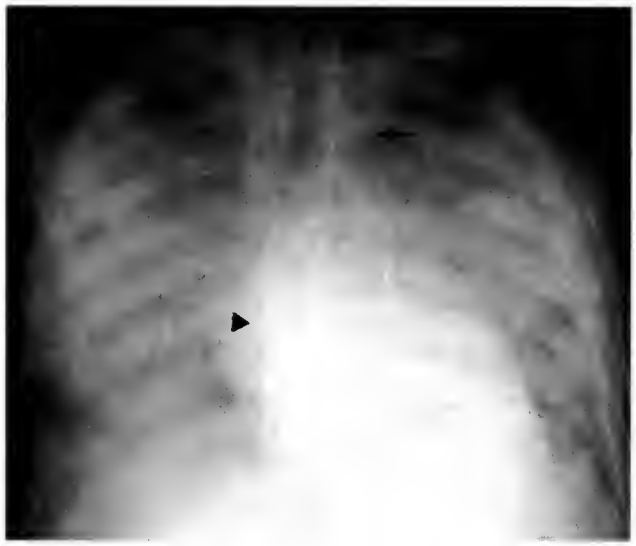

Fig. 12. Veno-arterial extracorporeal life support in a 19-year-old man with diffuse bilateral alveolar consolidation acute respiratory distress syndrome. Note the position of the right carotid arterial cannula with tip at the top of the aortic arch (arrow), and right internal jugular cannula with tip in the superior vena cava (arrowhead).

dial oxygen requirements. Inflation and deflation are linked to the ECG. The only radiopaque portion of the IABP is the tip, which should be located in the proximal descending thoracic aorta, at the inferior aspect of the aortic knob (see Fig. 11). If the tip is too proximal, the IABP may extend into the great vessels arising from the aortic arch, causing injury or obstruction of the left subclavian artery or occlusion/embolization of the left vertebral artery.[33] When positioned too distal in the descending aorta, it loses effectiveness and may overlie the ostia of the renal arteries, with the potential for renal embolism, or interfere with arterial supply to the bowel, resulting in ischemia.[34]

Advanced Cardiovascular Support Devices

Extracorporeal Life Support. Extracorporeal life support (ECLS) is a simple form of cardiopulmonary bypass, instituted at the bedside. It is used primarily in treating neonatal respiratory distress, and, more recently, in adults with acute, severe, and potentially reversible respiratory failure and otherwise high risk of mortality.[35] Deoxygenated blood from the venous system undergoes extracorporeal oxygenation and is returned to either the arterial (veno-arterial or V-A) or the venous (veno-venous or V-V) system. Arterial access is through the right carotid artery or a

Fig. 13. HeartMate left ventricular assist device in a 52-year-old man with a dilated cardiomyopathy in cardiogenic shock and awaiting heart transplantation. Note the opaque proximal portions of the efferent (arrows) and afferent (arrowheads) cannulas connecting to the ascending aorta and left ventricle respectively. A portion of the efferent component that connects what is seen here to the ascending aorta is not radiopaque,

common femoral artery; venous access is through the right internal jugular vein, or, less commonly, a femoral vein. The venous cannula tip should be in the distal SVC or right atrium, and the arterial cannula at the top of the aortic arch or distal innominate artery. Bear in mind that some cannulae have a 1–2 cm nonradiopaque extension at the tip, so the cannula tip is not where it appears to be (Fig. 12). Complications particular to ECLS are predominantly related to bleeding, particularly hemothorax, as these patients are anticoagulated and have chest tubes secondary to barotrauma.

HeartMate. The pneumatically-driven HeartMate device (Thermocardiosystems Inc, Woburn, Massachusetts) is the first implantable cardiac-assist device to gain U.S. Food and Drug Administration approval for commercial sale in the United States.[36] The pumping chamber, with afferent and efferent cannulae, has a unique radiographic appearance (Fig. 13). The HeartMate has a significant advantage over other devices in that the surfaces of the pumping chamber are textured, thus promoting formation of an adherent tissue lining derived from the patient's own blood. This greatly reduces the complication of thromboembolism and the need for long-term anticoagulation.[37] The pumping of blood is accomplished by the cyclical inflation of a polyurethane sac adjacent to a blood reservoir. A connecting transabdominal pneumatic tube connects to the drive console, which rests on a mobile cart. A newer version of the device is battery-powered and is small enough to be carried using a shoulder strap. The results of the first patients using the device have been encouraging: there has been a marked decrease in the incidence of thromboembolic complications.[36] Patients have been supported on the device for periods well over one year while awaiting transplantation.[38]

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Chest Imaging in the Trauma Intensive Care Unit

Jannette Collins MD MEd

Introduction
Aortic Injury
Lung Parenchymal Injury
Tracheobronchial Injury
Diaphragm Rupture
Injuries to the Bony Thorax
Pleural Manifestations of Chest Trauma
Cardiac Trauma
Soft Tissue Injuries of the Chest Wall
Esophageal Injury
Role of CT in Chest Trauma

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Introduction

In the United States, over 300,000 patients are hospitalized annually because of chest injuries.[1] Thoracic injury accounts for 25% of all traumatic deaths, and significant chest trauma is present in 50% of fatal traffic accidents.[2] Most of the chest trauma seen in civilian populations is blunt chest trauma (90%), usually the result of motor vehicle accidents and falls.[3]

A chest radiograph is usually the first imaging study obtained in the evaluation of trauma patients. This study is often compromised by limited exposure capability, expiratory views, poor or absent patient cooperation, suboptimal patient positioning, and magnification and distortion of the mediastinum on the supine chest radiograph. Studies have shown computed tomography (CT) to be superior to supine chest radiography in showing findings of chest trauma, although these CT findings do not always result in a change in patient management.[4–8]

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Aortic Injury

Traumatic rupture of the aorta accounts for 16% of fatalities resulting from motor vehicle accidents, and 85–90% of patients with traumatic aortic rupture die before reaching a medical facility.[9] In clinical series, 90% of aortic ruptures occur at the aortic isthmus (Fig. 1), just distal to the origin of the left subclavian artery.[10–13] Chest radiographic signs of aortic injury lack sensitivity and specificity (Table 1). No single or combination of radiographic signs is sufficiently sensitive to indicate all cases of traumatic aortic rupture on plain chest radiographs without a large number of aortographically negative studies.[14]

In some institutions, CT scanning has replaced conventional aortography in evaluating for aortic injury. Five millimeter contrast-enhanced CT scans can help exclude mediastinal hemorrhage and reduce the angiography rate in low-to-moderate-risk patients with a widened or indeterminate mediastinum on chest radiography.[15] However, a review of studies shows that a negative conventional CT scan does not exclude the possibility of an aortic laceration.[16–20] CT aortography can supplement conventional CT scanning and replace transcatheter aortography, except for small tears or indeterminate studies.[21]

The CT aortography protocol used at the University of Wisconsin consists of spiral scanning beginning at the top of the diaphragm and extending up through the aorta, with a collimation of 5 mm, a pitch of 2:1, and 3 mm reconstruction after administration of 150 mL of nonionic intravenous

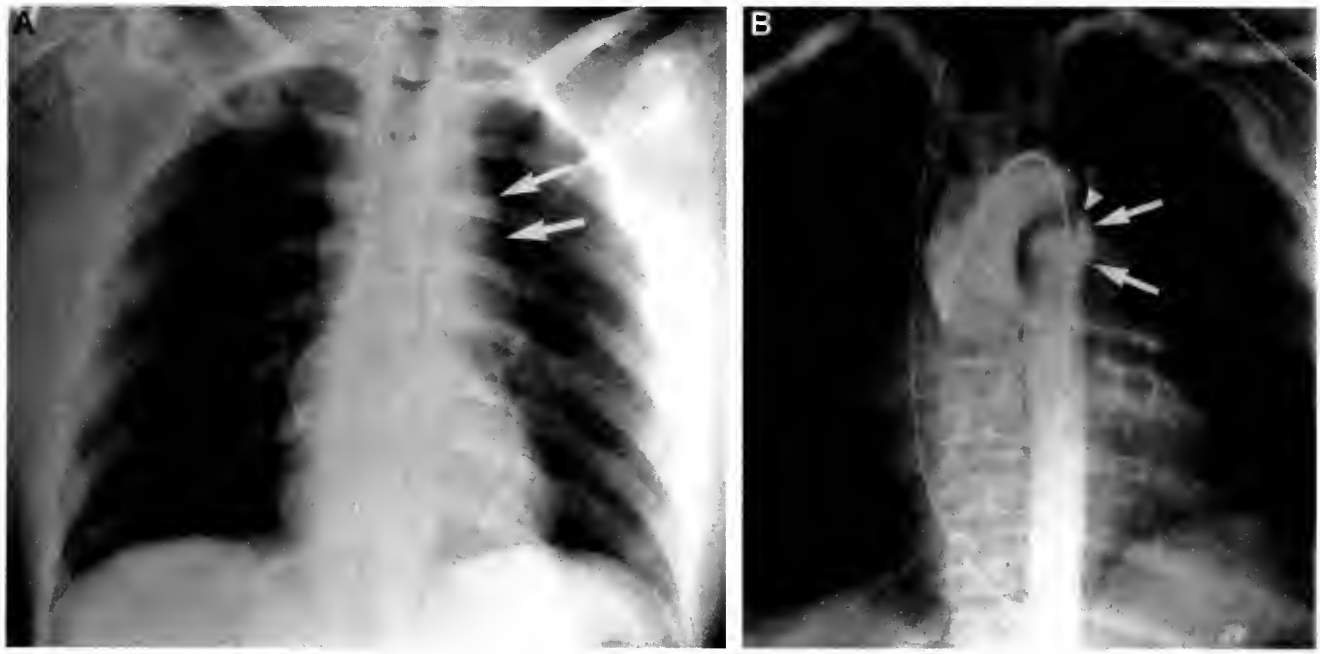

Fig. 1. Traumatic aortic laceration in an adult man after a motor vehicle accident. A: Anteroposterior supine chest radiograph shows widening of the mediastinum, with loss of the normal contour of the aortic arch (arrows). No other signs of aortic injury are present. B: Conventional aortogram shows a radiolucency representing laceration of the aorta at the isthmus (arrowhead), and pseudoaneurysm formation (arrows).

Table 1. Chest Radiograph Signs of Traumatic Aortic Injury

- Widening of the mediastinum
- Blurring of the aortic knob or contour
- Hemothorax
- Rib fractures
- Tracheal shift to the right
- Left apical cap
- Depression of the left main-stem bronchus below 40 degrees
- Nasogastric tube displacement to the right
- Pneumothorax
- Pulmonary contusion
- Widened left paraspinous line

contrast. If mediastinal hemorrhage is present, unless minimal and not centered around the aorta, without any direct signs of aortic injury, and no other explanation for the hemorrhage shows on CT, the patient is generally referred for conventional angiography. If direct signs of aortic injury are confirmed on CT, including (1) aortic caliber change at the site of injury, (2) focal outpouching of contrast consistent with pseudoaneurysm (Figs. 2 and 3), and (3) intraluminal flap, the surgeon determines whether to proceed to confirmatory conventional angiography.

Not only is CT useful for detecting direct signs of aortic injury, it can also show other causes of a wide mediastinum, including excessive mediastinal fat, adjacent lung contusion (Fig. 4), artifact due to supine positioning, vascular tortuosity, or persistent left-sided superior vena cava.[22] In experienced hands, transesophageal echocardiography has a reported sensitivity of 100% and specificity of 98% for diagnosing acute aortic injury, and in some institutions it is the diagnostic test of choice.[3]

Lung Parenchymal Injury

Abnormal lung parenchymal opacification in trauma patients can result from atelectasis, aspiration, edema, pneumonia, contusion, laceration, and fat embolization, and its etiology is commonly multifactorial. Pulmonary contusion results in leakage of blood and edema fluid into the interstitial and alveolar spaces. Pulmonary laceration, a more severe injury, results in disruption of the lung architecture.

CT is more sensitive than radiography in demonstrating contusions and lacerations.[5,7,8,23–27] On both chest radiographs and chest CT, pulmonary contusions present as areas of consolidation and/or ground glass opacification that tend to be peripheral, nonsegmental, and geographic in distribution (Fig. 5). Contusions are evident at presentation or within 6 hours and resolve, often without permanent sequelae, within days to weeks (Fig. 6), whereas pulmonary laceration may initially be masked by coexistent contusion and other forms of chest injury on the initial radiograph or CT scan, and lacerations generally take weeks to months to resolve, sometimes with residual scarring. Lung laceration results in disruption of alveolar spaces and formation of a cavity, usually filled with blood or air, and

Fig. 2. Traumatic aortic laceration in a 26-year-old man after a motor vehicle accident. A: Anteroposterior supine chest radiograph shows widening of the mediastinum, and a bumpy contour to the aortic arch (arrows). There is elevation of the right diaphragm, secondary to atelectasis (no diaphragm injury was detected on computed tomography [CT] scanning). B: Nonhelical CT (10 mm collimation) with intravenous contrast shows a focal outpouching of contrast material from the aortic arch, consistent with pseudoaneurysm, a direct CT sign of aortic injury (arrows). Note bilateral pleural effusions. C: More inferiorly, CT shows linear lucencies within the descending aorta, representing the intimal flap of aortic laceration (arrowheads), another direct CT sign of aortic injury. The descending aorta is focally widened at this level.

with consequent production of hematoma or pneumatocele (Figs. 7 and 8).

Pneumatoceles and hematomas can be multiple (Fig. 9), but are more commonly isolated lesions. They can be very large, but are usually 2–5 cm in diameter. The CT diagnosis of lung laceration is based on the presence of a localized air collection within an area of consolidation after trauma.[25]

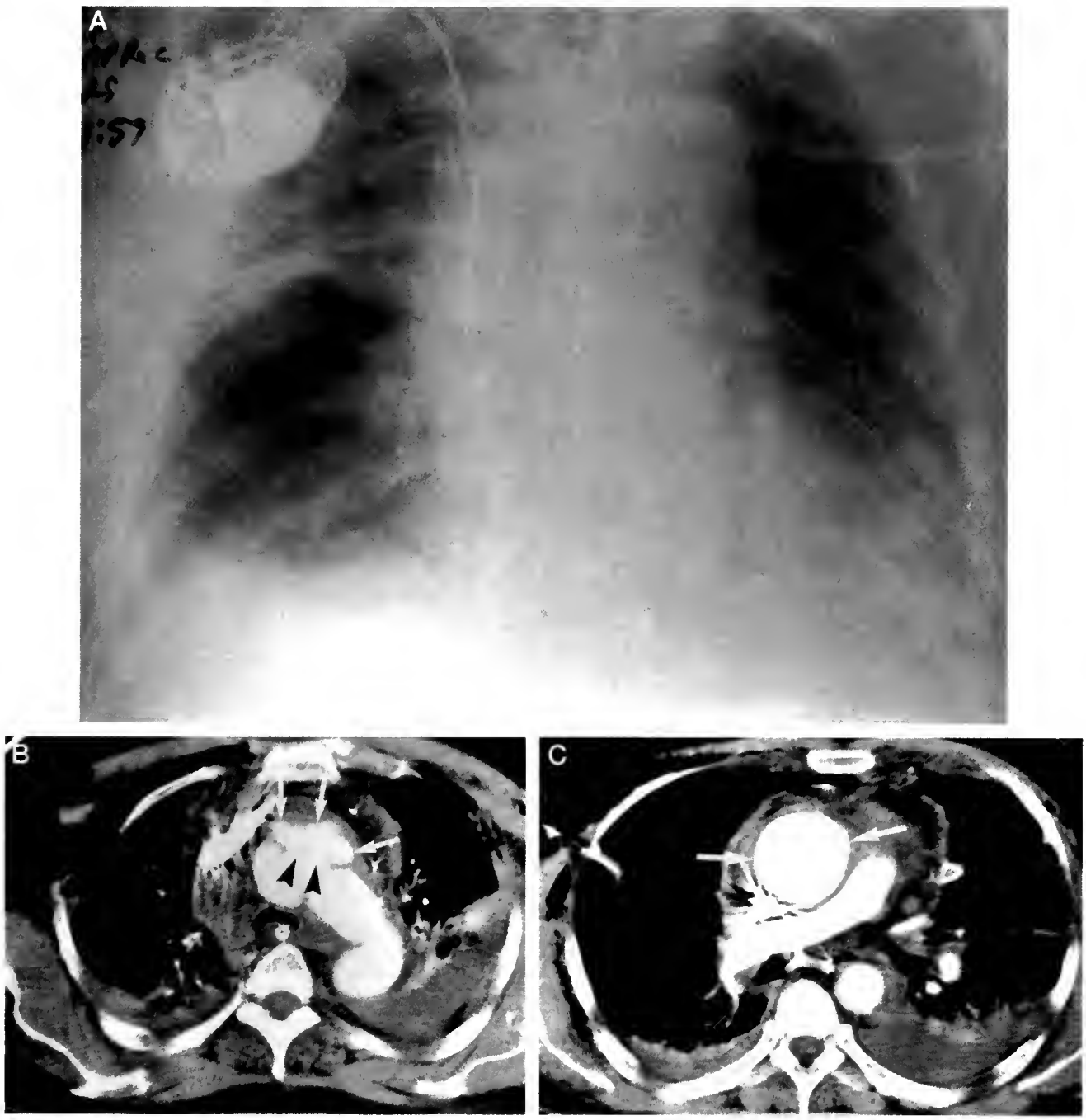

Fig. 3. Ruptured traumatic pseudoaneurysm of the aorta in an 80-year-old man after a high speed motor vehicle accident. A: Anteroposterior supine chest radiograph shows a wide mediastinum and bilateral scattered abnormal parenchymal opacities. A dual-lead pacemaker had been placed several years earlier for third degree heart block. There are bilateral chest tubes in place, and fractures of several right ribs. B: Helical CT (5 mm collimation) with intravenous contrast shows a pseudoaneurysm of the aortic arch (arrows), with a wide neck (arrowheads). There is high attenuation material surrounding the aorta, consistent with mediastinal hemorrhage from rupture of the pseudoaneurysm, and bilateral pleural effusions. C: More inferiorly, CT shows aneurysmal dilatation of the ascending aorta (arrows). The patient underwent surgical repair of the aortic arch and aortic valve replacement, but died one month after trauma.

Skeletal fractures, particularly those involving the long bones, can result in neutral fat droplets entering the bloodstream. These fat droplets become embedded in and occlude the vascular bed of the lungs and other organs. Hydrolysis of neutral fat by tissue lipase forms free fatty acids that have a toxic effect on the vascular endothelium and lung parenchyma, resulting in endothelial damage, increased capillary permeability, damage to alveolar lining

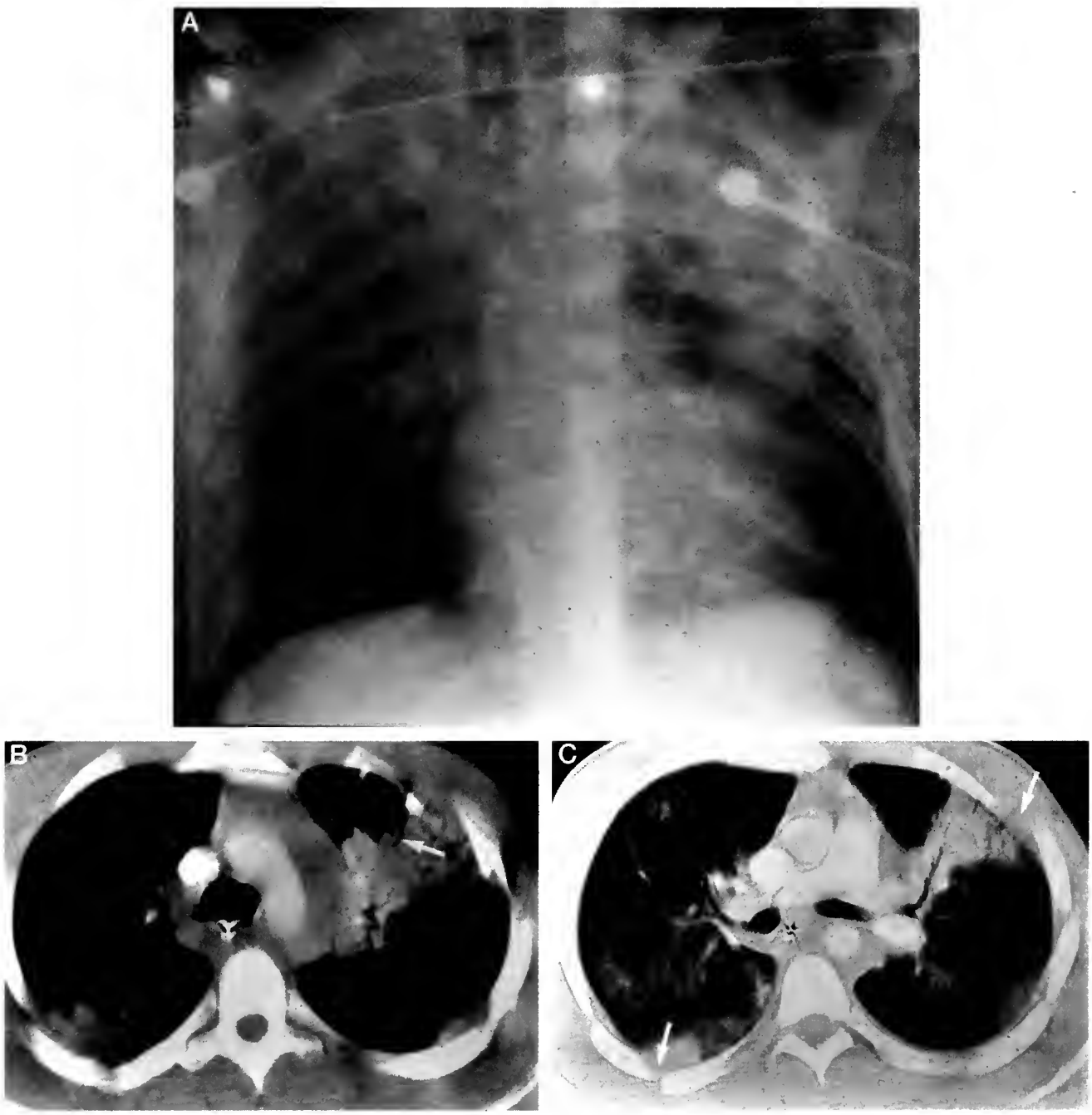

Fig. 4. Mediastinal hematoma in a 22-year-old man after a motor vehicle accident. A: Anteroposterior supine chest radiograph shows a widened mediastinum (partially obscured by the trauma backboard), a left apical cap, and consolidation of the left upper lobe. B: Helical computed tomography (CT) (7 mm collimation) with intravenous contrast shows high attenuation material surrounding the aorta (representing mediastinal blood), consolidation of the left upper lobe with air bronchograms (arrow), and patchy opacities in the superior segments of the lower lobes. C: More inferiorly, CT lung windows show left upper lobe consolidation, bilateral patchy opacities, and bilateral rib fractures (arrows). The aorta was normal on CT, but conventional aortogram was performed because of the mediastinal hematoma, and showed no aortic injury. The mediastinal hematoma is assumed to represent venous hemorrhage. The bilateral parenchymal opacities cleared within 10 days on follow-up CT, consistent with contusions.

cells, loss of surfactant activity, and the formation of hyaline membranes. Breakdown of platelets releases vasoactive substances, causing vasospasm and pulmonary capillary congestion. There is a latent period of 12–48 hours before fat embolization syndrome occurs, because of the time required to hydrolyze neutral fat and for vasculitis and pneumonitis to develop. Fat embolization "syndrome" refers to lung, central nervous system, and skin involve-

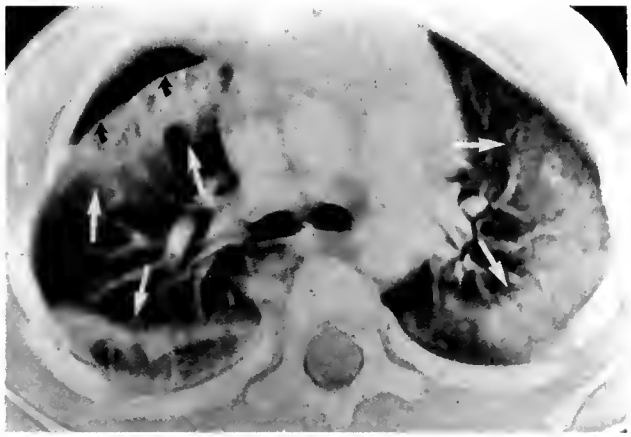

Fig. 5. Pulmonary contusions in a 4-year-old boy after a motor vehicle accident. Helical CT (5 mm collimation) shows a right pneumothorax (curved arrows) and bilateral peripheral, nonsegmental airspace opacities (straight arrows). Note bilateral rib fractures.

ment. The chest radiograph abnormalities are nonspecific (Fig. 10), occur after a 12–72 hour latent period, and include multiple focal alveolar opacities or a diffuse interstitial or alveolar pulmonary edema pattern.[28]

Tracheobronchial Injury

Tracheal or bronchial rupture can result from blunt or penetrating injury, or iatrogenically from intubation. The incidence of tracheobronchial injury (TBI) is reported as 0.4–1.5% in clinical series of major blunt trauma, and 2.8–5.4% in autopsy series of trauma victims.[29] Tracheobronchial rupture is associated with a 30% overall mortality rate, primarily from associated injuries.[30] There is no initial radiographic evidence of TBI in 10% of patients.[31] Failure to recognize TBI can result in death or allow cicatrization to occur, with airway obstruction arising days or months after initial injury. More than 80% of TBIs occur within 2.5 cm of the carina.[32,33] Rupture of the cervical trachea can occur as a "clothesline injury" when the neck is extended on high-speed contact with a rope, wire, or cable (often by individuals riding recreational vehicles or running), or when the neck of a driver strikes the top of a steering wheel, compressing the airway against the spine (Fig. 11).

Injury to the mediastinal trachea or major bronchi produces mediastinal emphysema that rapidly extends into the neck and face, and down to the shoulders and chest wall. A pneumothorax that does not resolve with functioning tube drainage is the sine qua non of mediastinal tracheal and major bronchial injury.[34] The "fallen lung sign,"[35] indicating bronchial tear, refers to the lung falling laterally and posteriorly in supine positioning, and inferiorly away from the hilum in the erect position, as a result of bronchial fracture allowing the lung to sag away from the

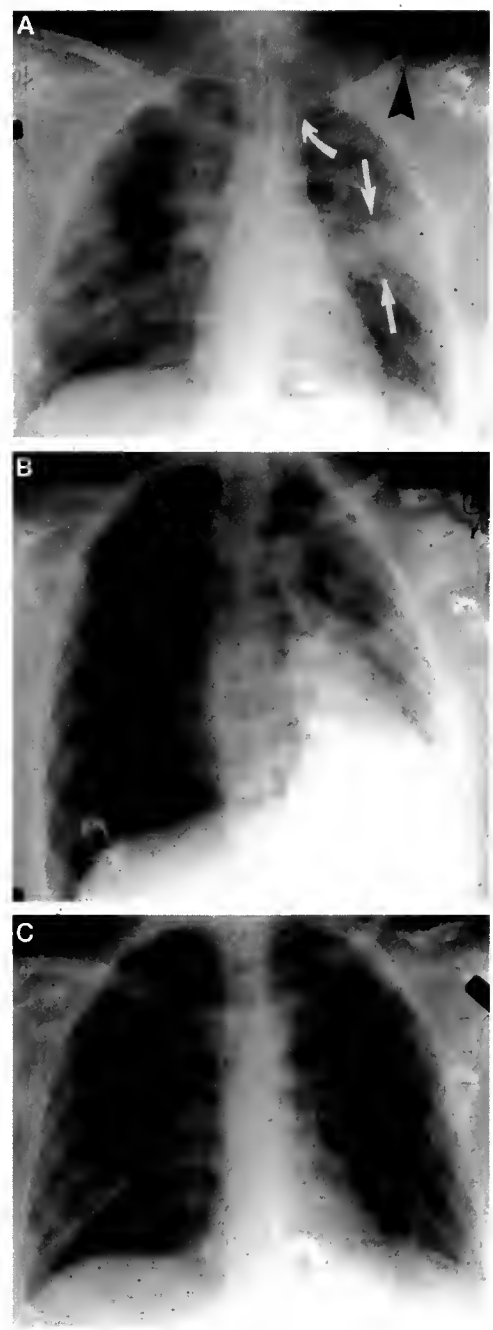

Fig. 6. Pulmonary contusions in a 21-year-old woman after a motor vehicle accident. A: Anteroposterior supine chest radiograph on day of trauma shows abnormal bilateral patchy parenchymal opacities, most notable in the left mid-lung (straight arrows). There is a displaced fracture of the left clavicle (arrowhead). The right subclavian venous catheter crosses the midline, and the tip is projected over the expected location of the left brachiocephalic vein (curved arrow). B: Two days later, upright anteroposterior chest radiograph shows a large right pneumothorax, left shift of the mediastinum, left lower lobe atelectasis, and elevation of the left diaphragm. The right venous catheter has been satisfactorily repositioned. C: Two days later, upright anteroposterior chest radiograph shows a small residual right pneumothorax after right chest tube placement, re-expansion of the left lower lobe, and clearing of the bilateral parenchymal opacities.

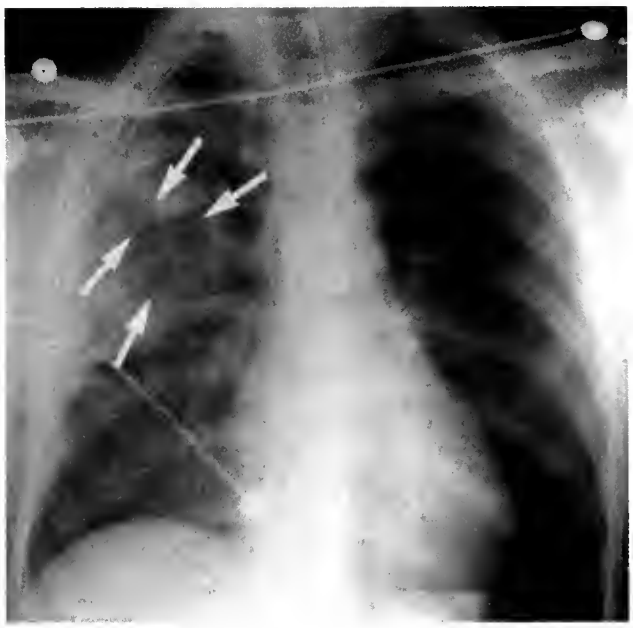

Fig. 7. Pneumatoceles in a young man after a motor vehicle accident. Anteroposterior supine chest radiograph shows multiple rounded lucencies within an area of consolidation in the right upper lobe (arrows). The presence of pneumatoceles implies pulmonary laceration, a more severe lung injury than pulmonary contusion.

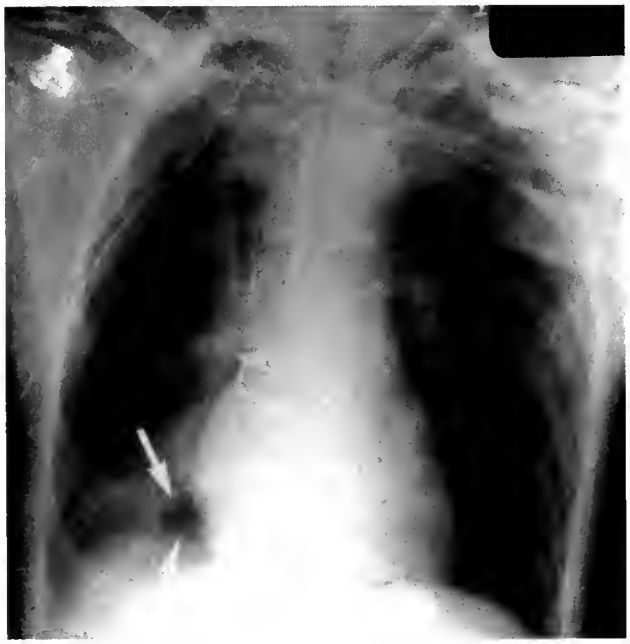

Fig. 8. Pneumatocele in a young man after a motor vehicle accident. Anteroposterior supine chest radiograph shows a triangular lucency within an area of consolidation in the right lower lobe (arrows). The right diaphragm is obscured due to atelectasis and laceration of the right lower lobe.

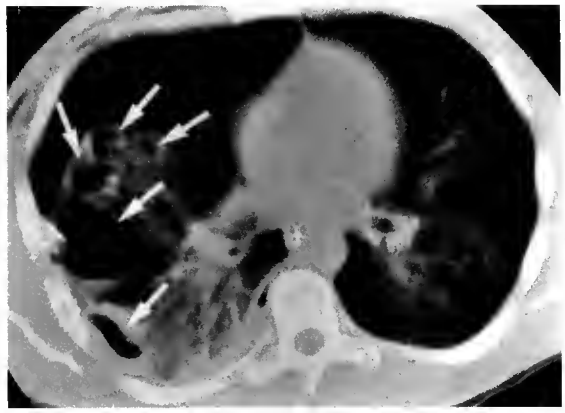

Fig. 9. Multiple pneumatoceles in a young man after a motor vehicle accident. Nonhelical CT (10 mm collimation) shows a right anterior pneumothorax, and numerous peripheral pneumatoceles (arrows). As pneumatoceles evolve, they typically develop thin walls.

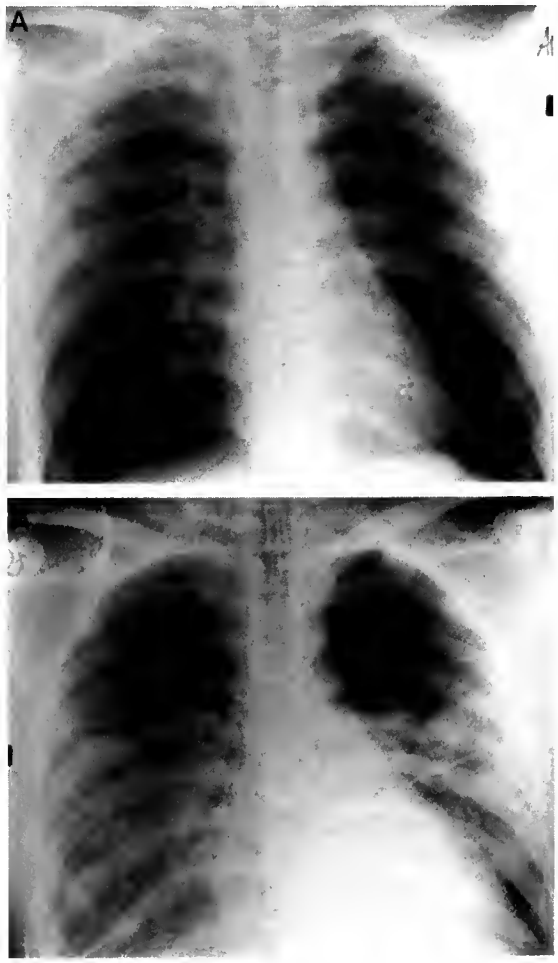

Fig. 10. Fat embolization syndrome in an 18-year-old woman after motorcycle accident, with fractures of both legs. A: Anteroposterior supine chest radiograph on day of trauma shows high positioning of endotracheal tube. The lungs are clear and there is no evidence of trauma to the chest. B: Anteroposterior supine chest radiograph 63 hours later shows new bilateral patchy abnormal air space opacities, mainly involving the mid-lung and lower lung zones. The patient also developed hypoxia, a petechial rash, and fat droplets within her blood.

Fig. 11. Traumatic tracheal tear in a 12-year-old girl after bicycle accident. The girl's neck was impaled against the metal bar of a dumpster. A: Nonhelical CT (5 mm collimation) shows an eccentric collection of air around the endotracheal tube, representing herniation of the endotracheal tube balloon through a posterolateral tear in the trachea (arrows). A long tracheal tear, from the second tracheal ring to the carina, was confirmed at surgery. There are bilateral pneumothoraces, abnormal mediastinal air, and massive subcutaneous emphysema. B: More inferiorly, CT shows pneumomediastinum, with air outlining the superior vena cava (curved arrows), aortic arch (long straight arrows), and azygos vein (short straight arrows). The abnormal air is outlined by the pleurae laterally (arrowheads).

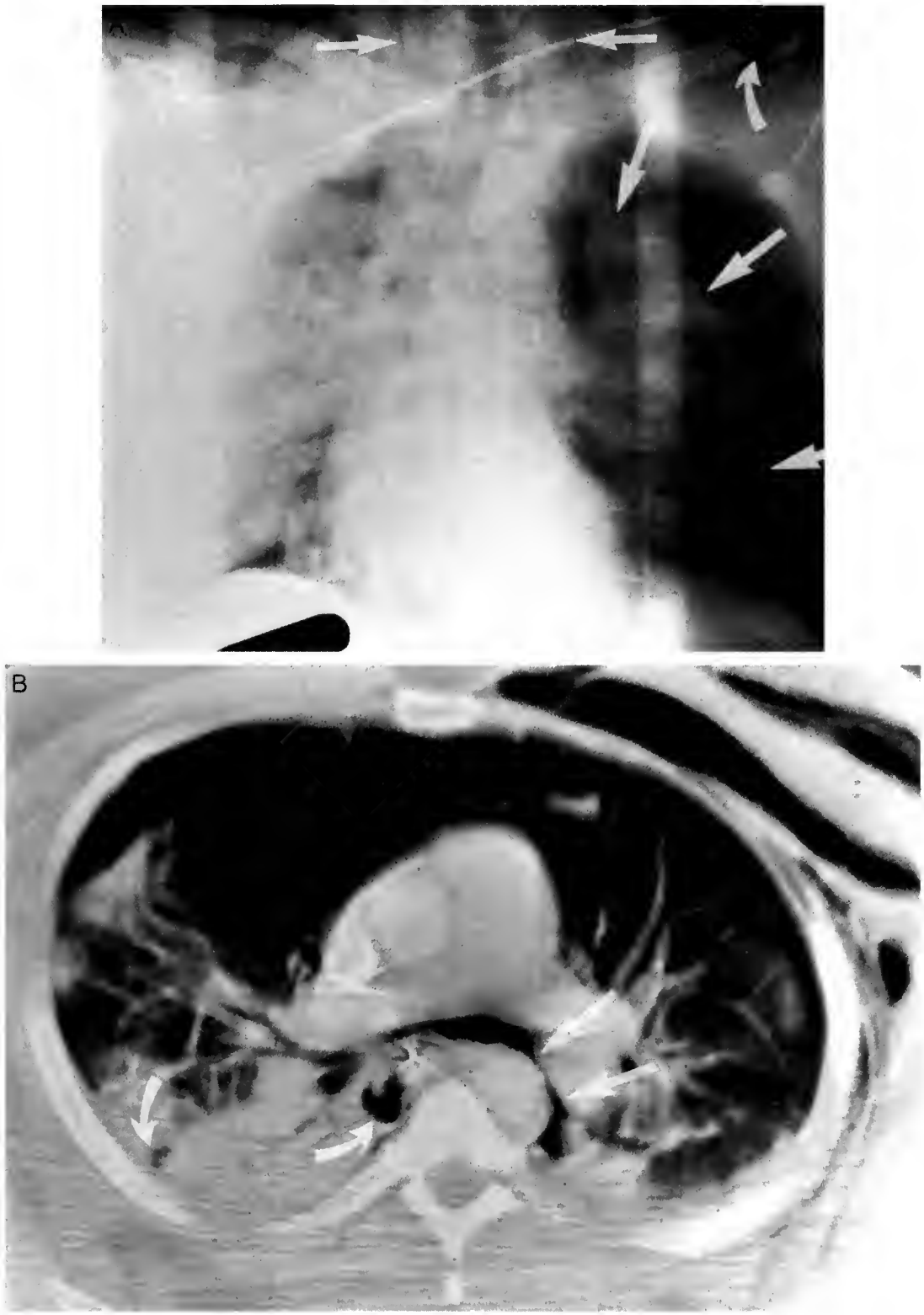

Fig. 12. Traumatic bronchial tear in a 24-year-old man after a motor vehicle accident. A: Anteroposterior supine chest radiograph shows a large left pneumothorax, causing right shift of the mediastinum, with the collapsed lung "falling out" laterally and posteriorly (long arrows) instead of collapsing completely toward the hilum. The left clavicle is fractured (curved arrow), and there is subcutaneous air within the neck bilaterally (short arrows). B: Nonhelical CT (10 mm collimation) shows abrupt tapering of the left main bronchus and air leaking from the torn bronchus posteriorly (long straight arrows). There are pneumatoceles within the right lung (curved arrows), in areas of lung laceration. There is subcutaneous emphysema of the left chest wall, a right pneumothorax, and pneumomediastinum.

Fig. 13. Traumatic tear of the bronchus intermedius in a young woman after a motor vehicle accident. A: Nonhelical CT (10 mm collimation) shows an abnormal air collection adjacent to the bronchus intermedius, extending posteriorly and communicating with the pleural space (arrows). There is a right pneumothorax, collapse of the right middle and lower lobes, and patchy areas of atelectasis and contusion in the left lower lobe. B: More inferiorly, CT shows the collapsed lung, surrounded by pneumothorax, which has "fallen" laterally and posteriorly away from the hilum (arrows).

Fig. 14. Traumatic rupture of the left diaphragm in a young man after a motor vehicle accident. Anteroposterior supine chest radiograph shows a large area of abnormal lucency within the left inferior chest (straight arrows), representing herniation of the stomach through a diaphragmatic tear. The stomach has mass effect, causing mediastinal shift to the right. The diagnosis is confirmed on this plain film by the presence of the superiorly positioned intragastric nasogastric tube (curved arrows). The mediastinum is widened and aortic tear was suspected, but aortogram was normal.

Table 2. Chest Radiograph Signs of Traumatic Rupture of the Diaphragm

- Displacement of stomach, colon, or small bowel into the thorax
- Superior displacement of an intragastric nasogastric tube
- Pleural effusion
- Basilar opacity causing inability to visualize the diaphragm
- Irregular or lumpy diaphragm contour
- Fractures of the lower ribs

hilum (Figs. 12 and 13). The vascular pedicle remains intact, and the lung remains perfused, although underventilated. In cases of pneumothorax without TBI, the lung collapses toward the hilum. An indication of tracheal tear, especially in conjunction with abnormal air in the soft tissues of the neck, is elevation of the hyoid bone above the level of C3 as seen on a lateral radiograph of the cervical spine.[36] This hyoid elevation is caused by injury to the infrahyoid musculature, which allows unopposed elevation by suprahyoid musculature. Another sign of tracheal transection is acute overdistention of the endotracheal tube cuff. In tracheal rupture, the balloon can herniate through the tear.[37]

Diaphragm Rupture

Acute diaphragmatic rupture occurs in 1–7% of patients following major blunt trauma,[38–40] and the diagnosis is missed on initial presentation in up to 66% of patients.[41–47] Between 75% and 95% of patients with acute diaphragm rupture have abnormal chest radiographs (Fig. 14), but only 17–40% have highly suggestive radiographic findings (Table 2).[48–51] Rupture of the right hemidiaphragm, once thought to account for only 10% of all diaphragm ruptures, probably occurs with almost the same frequency as rupture of the left hemidiaphragm.[51] If diaphragm rupture is not promptly diagnosed, the patient may remain asymptomatic or develop incarceration of herniated abdominal viscera, which can occur long after the incident of trauma.

Fig. 15. Multiple skeletal fractures in a 21-year-old man after a motor vehicle accident. A: Anteroposterior supine chest radiograph shows a widened mediastinum, bilateral patchy abnormal parenchymal opacities, and bilateral chest tubes. B: Nonhelical CT (10 mm collimation) shows an oblique fracture of the sternum (arrows), bilateral rib fractures, mediastinal hematoma, and bibasilar areas of consolidation with air bronchograms (arrowheads). The aorta was normal on CT and conventional aortogram. C: At the level of the base of the heart, CT shows a comminuted burst fracture of the thoracic spine, with fracture fragments displaced posteriorly into the spinal canal. There is blood surrounding the descending aorta within the retrocrural space. D: Lung windows show an anterior pneumothorax (arrows) and bibasilar consolidation. E: CT 9 days later shows clearing of left basilar consolidation, consistent with resolving contusion, and development of a pneumatocele at the right base (arrows), representing disruption of the alveolar spaces and formation of an air-filled cavity within an area of pulmonary laceration.

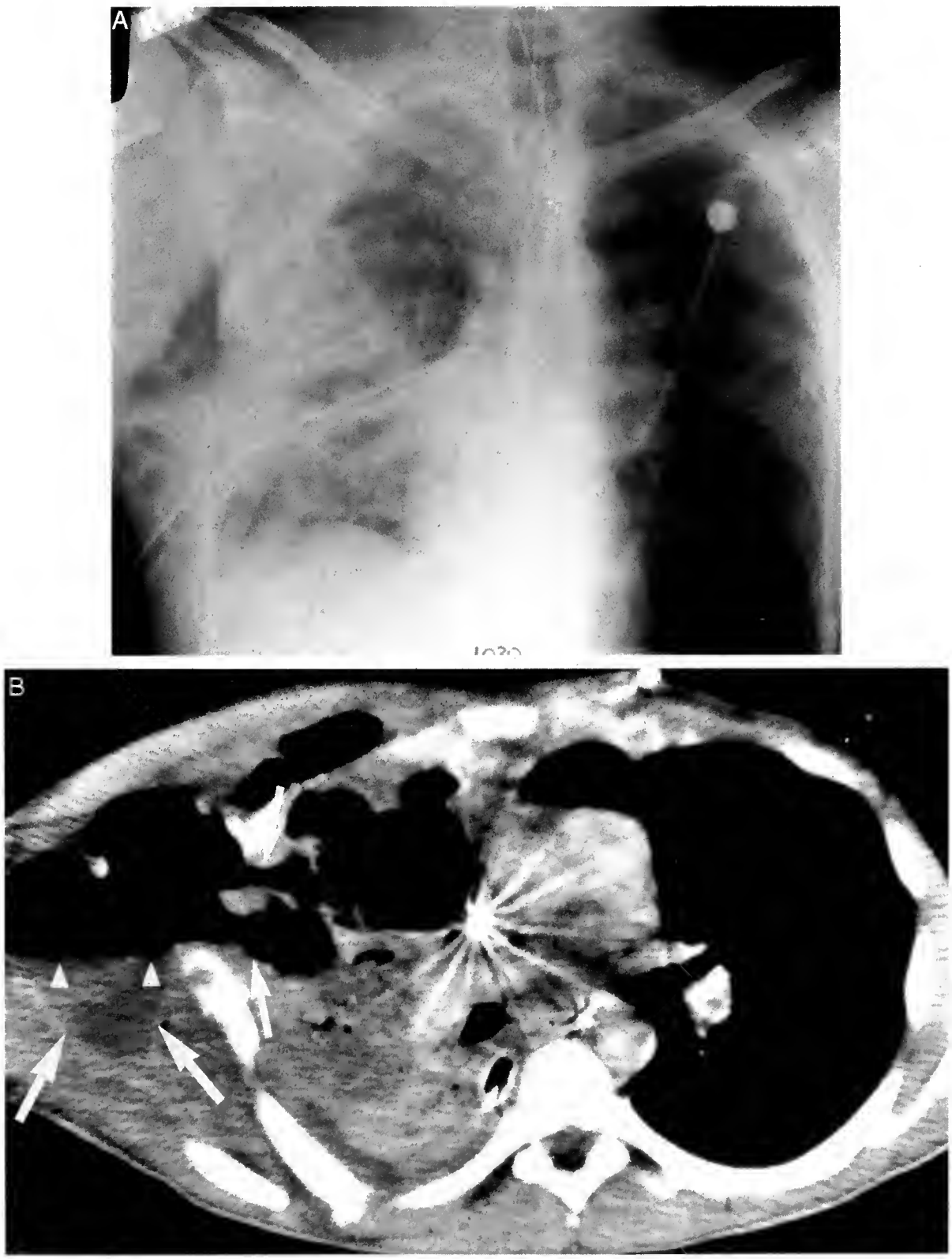

Fig. 16. Flail chest in a 29-year-old man injured by the steering wheel during a motor vehicle accident. A: Anteroposterior upright chest radiograph shows comminuted fractures of more than 3 adjacent ribs on the right, and a large collection of air in the right subcutaneous tissues. There is associated atelectasis and lung injury, and a right basilar chest tube. B: Nonhelical CT (10 mm collimation) with intravenous contrast shows displaced rib fracture fragments, diffuse lung injury, and a bronchopleural cutaneous fistula (short arrows) leading to a large hematoma within the right chest wall (long arrows) containing an air/fluid level (arrowheads).

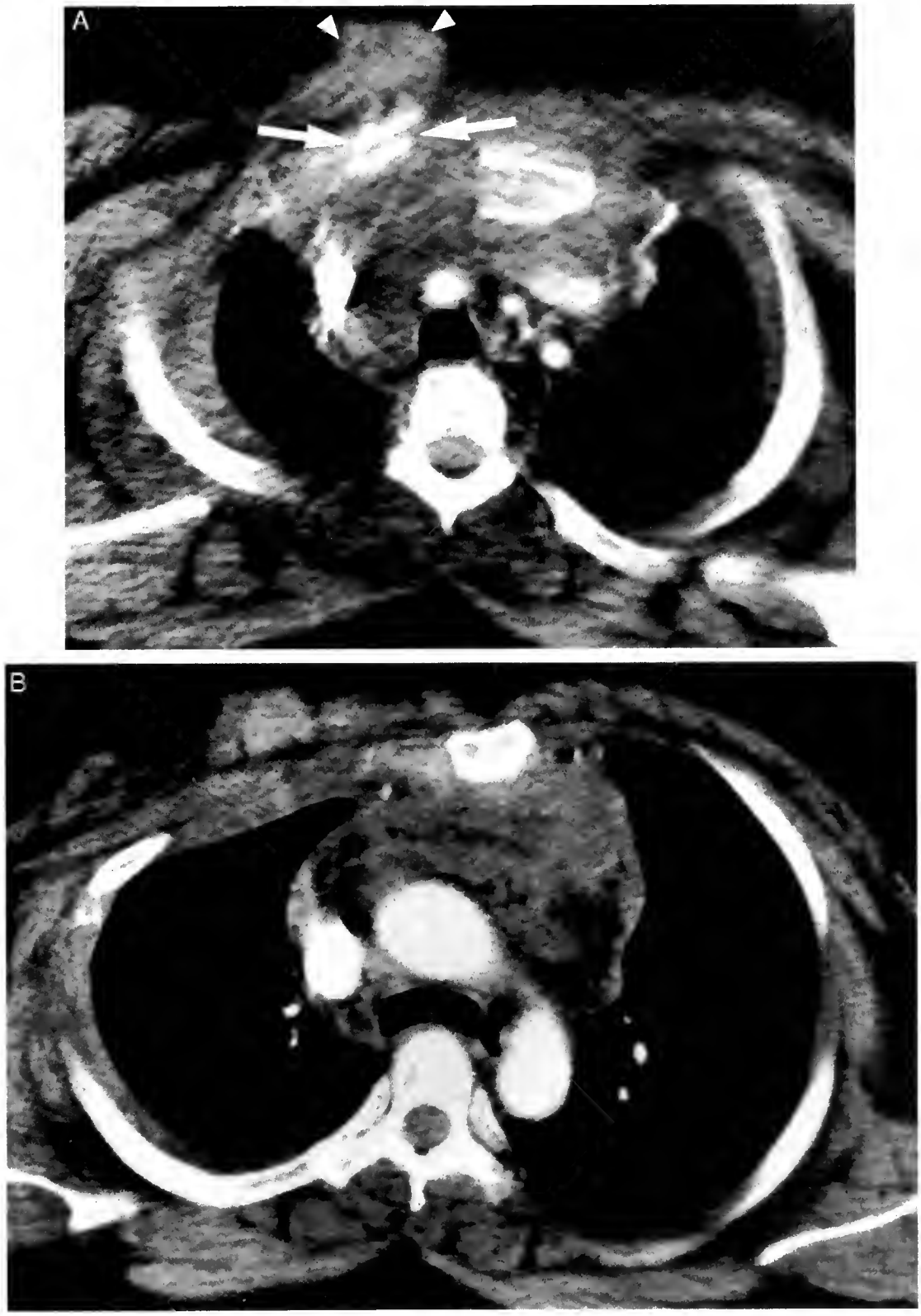

Fig. 17. Displaced sternal fracture in a 43-year-old man after a motor vehicle accident. A: Helical CT (5 mm collimation) with intravenous contrast shows a fracture of the sternum with anterolateral displacement of the right fracture fragment (arrows), mediastinal hemorrhage, and right hemothorax. There is a hematoma of the anterior chest wall (arrowheads). The patient also had multiple right rib fractures and a fractured femur. No aortic injury was present on CT or conventional aortogram. B: More inferiorly, CT shows continuation of the mediastinal hematoma, caused by the sternal fracture.

Fig. 18. Traumatic sternoclavicular dislocation in a young man after a motor vehicle accident. Nonhelical CT (5 mm collimation) without intravenous contrast shows posterior displacement of the left clavicle (short straight arrows). Note proximity of displaced clavicle to left brachiocephalic vein (arrowhead), left common carotid artery (long straight arrow), and left subclavian artery (curved arrow).

Helical CT is useful in making the diagnosis of acute diaphragm rupture and is superior to conventional CT because volumetric data acquisition allows high-quality sagittal and coronal reformations. Individual diagnostic sensitivity for detecting diaphragmatic rupture is 54–73%, and specificity is 86–90%.[52] Direct CT findings associated with acute rupture include (1) diaphragmatic discontinuity, (2) intrathoracic herniation of abdominal contents, and (3) waist-like constriction of bowel ("collar sign"). Associated CT findings include liver laceration, hemoperitoneum, hemothorax, splenic laceration, renal contusion, lower lobe atelectasis, and lower rib fractures. There is a normal increase in diaphragmatic defects with age, not related to trauma.[53] When the diagnosis of diaphragm rupture remains uncertain after CT or for delayed, nonacute, clinical or radiologic presentation that suggests diaphragmatic injury, magnetic resonance imaging can be done with T1-weighted sagittal and coronal sequences.[54]

Injuries to the Bony Thorax

Significant injury to ribs, clavicles, scapulae, sternum, and spine can occur as a result of blunt chest trauma. Thoracic spine fractures account for 16–30% of all spine fractures (Fig. 15), resulting in complete neurological deficits in 62% of patients.[55,56] A supine chest radiograph provides an opportunity to evaluate the thoracic spine, but optimal evaluation requires frontal and lateral collimated images. CT and magnetic resonance imaging are more helpful in distinguishing unstable burst fractures from stable, simple, anterior wedge compression fractures.[57] Fractures to the upper ribs, clavicle, and upper sternum are significant in that they are accompanied by brachial plexus or vascular injury in 3–15% of patients.[58] Fractures of the lower ribs should increase the suspicion of splenic, hepatic, or renal injury, which can be confirmed with CT. Double fractures of 3 or more adjacent ribs can result in paradoxical motion of a "flail" segment during the respiratory cycle, which can impair respiratory mechanics and result in atelectasis and pulmonary infection (Fig. 16). Rarely, a segment of lung can herniate through a defect in the chest wall created by a flail segment. The diagnosis may be made by plain radiographs, but is easier to detect by CT.[59] Sternal fractures occur in 8% of major thoracic trauma admissions (Fig. 17).[60] Sternal fracture may be associated with myocardial contusion, which is often clinically silent. Posterior dislocation of the clavicle can result in injury to the great vessels, superior mediastinal nerves, trachea, and esophagus. Although sternoclavicular dislocations are demonstrable using angled chest radiographs, they are more easily detected by CT (Fig. 18).[59]

According to one study,[61] scapular fractures are diagnosed on the initial chest radiograph in only 57% of patients, (Fig. 19). In the group of patients in which the fractures were not recognized, the fracture was visible and overlooked in 72%, not included on the examination in 19%, and was obscured by superimposed structures or artifacts in 9%. CT of the chest should reveal most scapular fractures, especially if utilized in combination with plain films. Scapulothoracic dissociation is a rare injury characterized by lateral displacement of the entire forequarter with intact overlying skin, complete acromioclavicular separation, and usually multiple fractures of the ipsilateral upper extremity, and is usually accompanied by avulsion injuries of the brachial plexus and subclavian nerves.[62]

Pleural Manifestations of Chest Trauma

Pneumothorax, seen radiographically, occurs in almost 40% of patients with blunt chest trauma.[63,64] All pneumothoraces in trauma patients should be considered potentially life-threatening. Even small pneumothoraces can rapidly enlarge and become clinically significant with positive pressure ventilation or general anesthesia. CT is much more sensitive for diagnosing pneumothorax in the supine patient than is chest radiography.[65,66] CT scanning identifies pneumothoraces that cannot be seen on conventional supine radiographs in 10–50% of patients with blunt trauma to the chest (Fig. 20).[65–67]

Pleural effusions developing in the acute posttraumatic setting usually represent hemothorax, and a rapidly expanding pleural effusion is usually a result of arterial bleeding. CT can be helpful in distinguishing intrapleural hematoma from other pleural collections by showing the

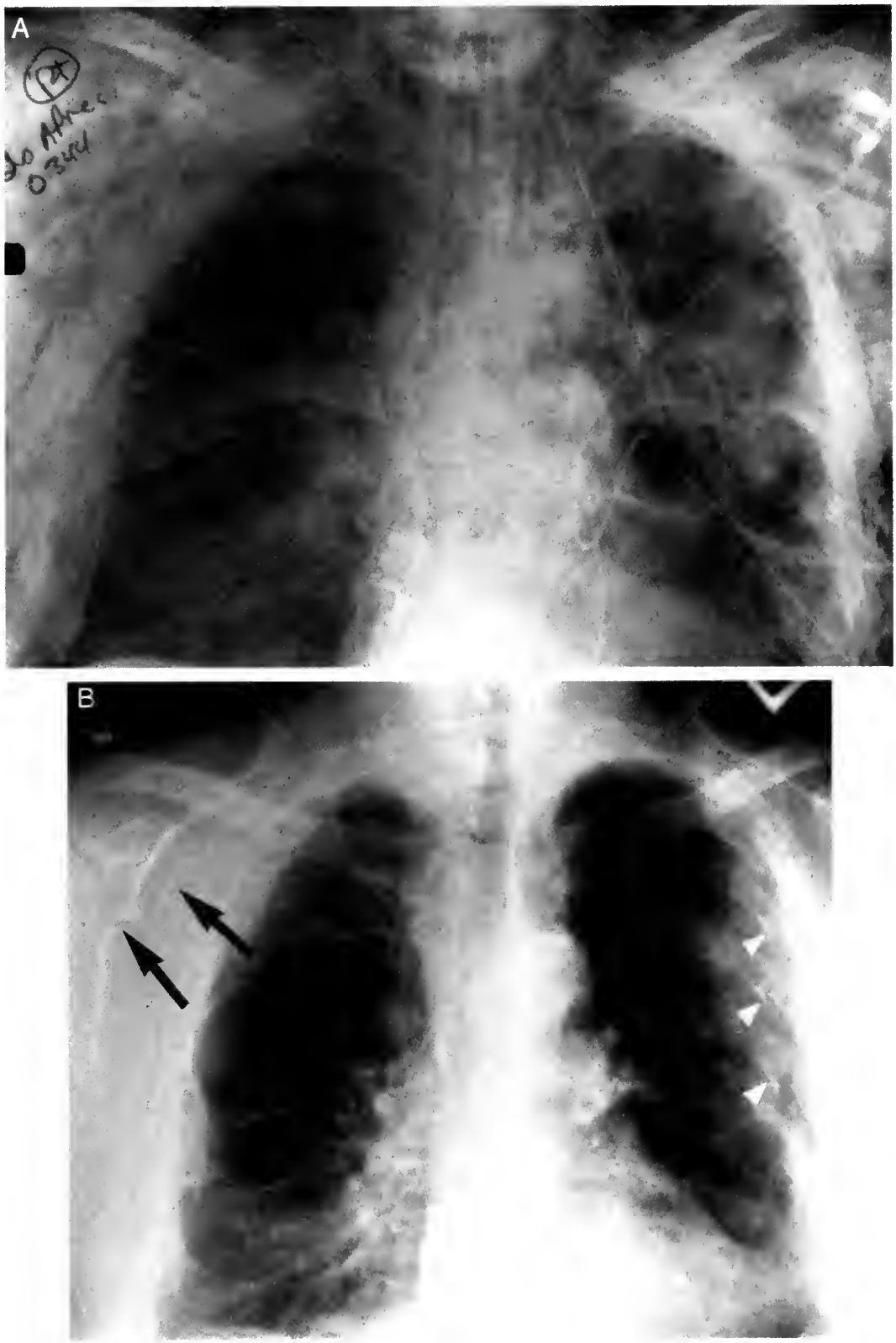

Fig. 19. Scapular fracture in a 62-year-old man after a motor vehicle accident. A: Anteroposterior supine chest radiograph on the day of trauma shows massive bilateral subcutaneous emphysema, which obscures the bones and lung parenchyma. There are 2 chest tubes in each chest, and no definite pneumothorax. Fractures of the right ribs are visible. B: Anteroposterior supine chest radiograph 10 days later, after resolution of subcutaneous emphysema, shows numerous contiguous right posterolateral rib fractures, consistent with flail chest, and a comminuted fracture of the right scapula (arrows). There are also numerous left rib fractures (arrowheads).

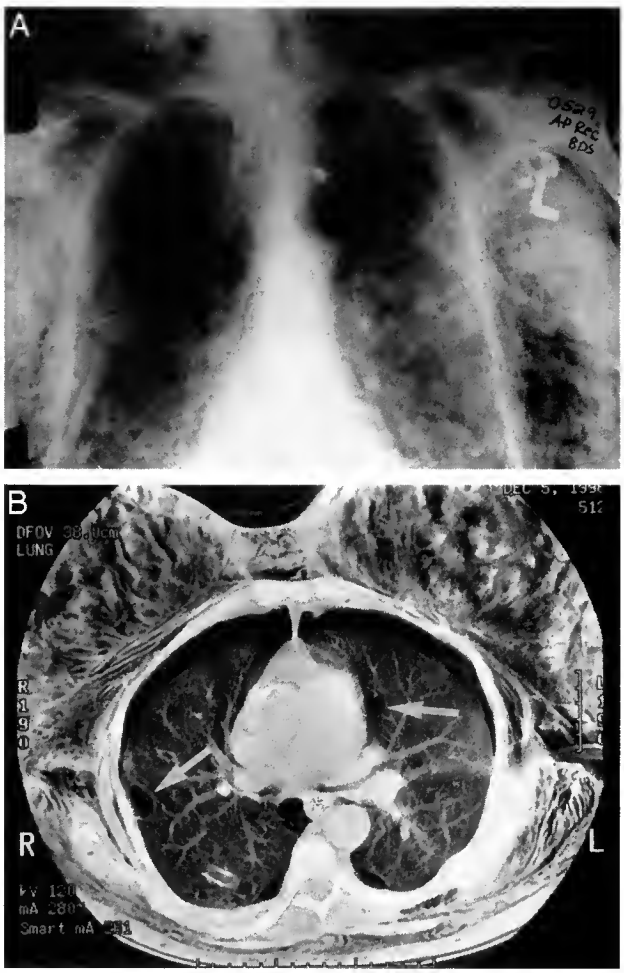

Fig. 20. Subcutaneous air obscuring lung and pleural disease in a 61-year-old woman after a motor vehicle accident. A: Anteroposterior supine chest radiograph on the day of trauma shows massive subcutaneous air bilaterally, compromising evaluation of the pleura, lung parenchyma, and bones. No definite pneumothorax is seen. Note bilateral chest tubes. B: Nonhelical CT (10 mm collimation) allows evaluation of the lung parenchyma, which is not superimposed under the emphysematous chest wall. There are bilateral pneumothoraces, and multiple bullae (arrows).

high CT attenuation of blood (Fig. 21).[68] CT is superior to chest radiography in distinguishing pleural fluid from other causes of abnormal radiographic density such as atelectasis, parenchymal injury, or pneumonia, and can show loculation of pleural fluid and better delineate complex pleuroparenchymal opacities.

Cardiac Trauma

The heart and pericardium are well-protected from nonpenetrating injury, although numerous traumatic injuries can occur (Table 3). Rapid accumulation of blood in the pericardial space can result in cardiac tamponade and severe hemodynamic compromise. Bedside sonographic evaluation of the heart is the study of choice to quickly and noninvasively detect pericardial fluid. CT scanning will show pericardial hemorrhage as having high CT attenuation. Associated CT scan findings of cardiac tamponade include distention of the inferior vena cava, hepatic, and renal veins, and periportal edema within the liver.[59] Pericardial rupture is a rare consequence of thoracic trauma, with an incidence of only 0.11% reported in one series.[69] The diagnosis can be made with chest radiography or CT when there is herniation of air-containing abdominal viscera into the pericardium accompanying diaphragmatic rupture. Pneumopericardium can occur when air enters through a pericardial disruption in the presence of pneumothorax.

Cardiac contusion can result from blunt chest trauma in 8–76% of patients.[70,71] The diagnosis is usually made from electrocardiography, nuclear cardiac imaging, or echocardiography. The right ventricle is most frequently injured, as it comprises almost 3 times more exposed anterior surface of the heart than does the left ventricle.[59] Chest radiography and CT scanning may show evidence of congestive heart failure, ventricular aneurysm, or massive cardiac enlargement.

Soft Tissue Injuries of the Chest Wall

CT scanning can easily distinguish chest wall from parenchymal or mediastinal injury, whereas this differentiation may not be possible with chest radiography. Soft tissue hematomas of the chest wall are readily distinguished from parenchymal injury, and subcutaneous emphysema will not be confused with pneumothorax on CT scanning.

Esophageal Injury

Esophageal disruption can occur from crushing of the esophagus between the spine and trachea, traction from hyperextension, and direct penetration by cervical spine fracture fragments.[72] Rupture of the esophagus by blunt external trauma is rare, comprising only 10% of all cases.[73] Chest radiography may show persistent severe pneumomediastinum or pneumothorax, left pleural effusion, a widened paraspinal line, and retrocardiac opacification. CT scanning can show similar findings, in addition to leakage of oral contrast from the disrupted esophagus into the mediastinum or pleural space, and changes of mediastinitis.

Role of CT in Chest Trauma

CT scanning is superior to chest radiography in demonstrating pneumothoraces, hemothoraces, pulmonary contusions, and fractures. CT has obviated the need for conventional aortography in a number of patients, and CT aortography may become the new gold standard for the

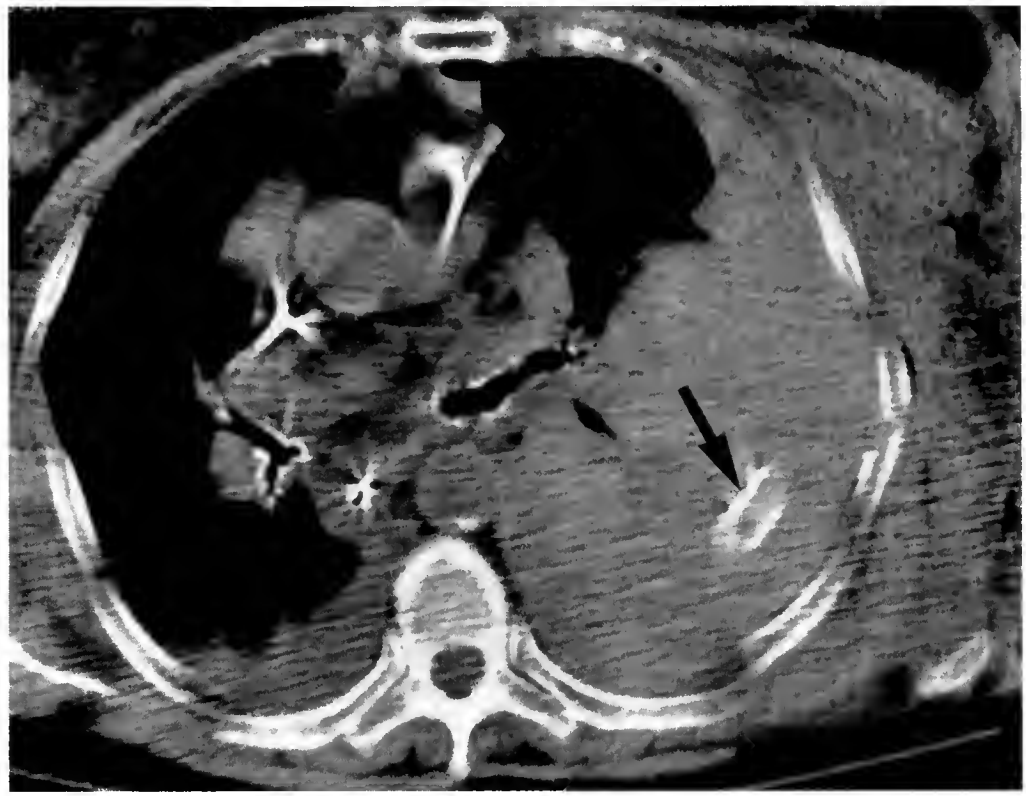

Fig. 21. Traumatic left hemothorax in a 78-year-old woman after a motor vehicle accident. Helical CT (7 mm collimation) without intravenous contrast shows a large intrapleural high-attenuation fluid collection on the left. Bloody fluid was drained through the left chest tube (arrow). The hemothorax has mass effect, and causes right shift of the mediastinum. There is a smaller right pleural effusion. Note hematoma and subcutaneous emphysema of the left chest wall.

Table 3. Traumatic Injuries to the Heart and Pericardium

- Myocardial contusion or infarction
- Myocardial laceration leading to hemopericardium and tamponade
- Septal defect
- Myocardial aneurysm
- Coronary artery damage
- Chordae rupture with valve insufficiency
- Pneumopericardium
- Postpericardiectomy syndrome and constrictive pericarditis (late sequelae)
- Herniation of the heart through a pericardial tear

diagnosis of acute aortic injury. Further studies on a larger population of trauma patients are required to determine the sensitivity and specificity of CT for this diagnosis. CT scanning provides a look at the entire chest, in addition to the aorta, which is a distinct advantage over transesophageal echocardiography or conventional aortography. In addition to showing fractures, CT also shows related soft tissue injuries, such as great vessel injury from fracture-dislocation of the clavicle, and splenic laceration from adjacent rib fractures. Chest CT could be quickly performed on all trauma patients referred for abdominal CT, as a way of detecting serious chest injuries early.

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Chest Imaging in the Neurosciences Intensive Care Unit

Daniel A Henry MD

Introduction
The Neurovascular Axis
Neurogenic Pulmonary Edema
Intravascular Therapy
The Neurosciences Intensive Care Unit
Neurological Deficits Impacting Cardiorespiratory Function
Sequelae of Care
Postoperative Situations and Attendant Issues
Summary
[Respir Care 1999;44(9):1064–1077] *Key words: thoracic imaging, pulmonary disease, intensive care unit imaging, neuro-pulmonary disorders, neurogenic edema, neuro-pulmonary complications.*

Introduction

Intensive care has evolved over the last several years from treatment areas originally intended to support patients requiring acute ventilatory management[1] to system-specific or organ-specific, clinically-focused critical care units designed to bring together highly skilled personnel and services to serve specific patient populations. Patients are admitted to intensive care units (ICUs) because of the complexity or severity of their illnesses, requirements for respiratory or cardiovascular support, the need for special monitoring or nursing care, risk of instability, or combined needs for care that exceed the standard level available in non-ICU environments.[2,3] These demands, coupled with advances in medical technology and refinements in subspecialty care and training, have spawned burn units, bone marrow transplant units, shock-trauma units, and neonatal ICUs, among others. These highly clinically segregated units are in addition to the "standard" ICUs for patients requiring medical respiratory or general medical, coronary medical, cardiac surgical, pediatric, or general surgical care. Aided by significant advances in the understanding of brain injury, the substantial progress in treatment options for neurological conditions, and technologic improvements in therapeutic and monitoring devices, neurological critical care has evolved into the neurosciences ICU (NSICU), providing integrated care for both neurosurgical and neurological patients.

The NSICU hosts patients with head injury, acute ischemic stroke, aneurysmal subarachnoid hemorrhage, seizures, spinal cord injury, acute neuromuscular ventilatory failure, and other conditions, and patients following both spinal and cranial neurosurgical procedures. Many patients requiring intensive care have similar clinical problems and conditions, regardless of their original diagnosis or the ICU in which they reside, but there are also significant differences in presentation, therapeutic appliances, and treatment focus and options.

Portable chest radiography and the portable chest radiograph have become indispensable tools in the care of the ICU patient. Efficacy studies have shown that a significant percentage of these portable studies have documented an unexpected finding, many of which require a change in patient management or modification of a catheter or tube position.[4] Portable chest radiography is a relatively inexpensive, portable, reproducible method of acquiring unique information regarding the patient's respiratory and cardiovascular condition, as well as the status of radiographically visible support appliances. Prompt availability of service, consistent image quality, and timely interpretation are required to provide optimal care.

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As in other ICU settings, portable chest radiography is instrumental in evaluating cardiac function, fluid balance and lung water, airway maintenance and ventilation, air or fluid collections, support devices, and the development of thoracic complications. The focus will change somewhat for postoperative patients. Some radiographic findings portray differing clinical situations from one ICU to another; findings of radiographic concern in one ICU setting may be expected or desired outcomes in another. Thus, there is a need to understand the nuances of the various clinical conditions and their focused care as they relate to interpretation of the portable chest radiograph. Therefore, the present discussion focuses on the chest radiographic correlations of the neurovascular axis and measures employed to address the primary intracranial event, developing neurologic deficits impacting cardiorespiratory function, sequelae of care, postoperative situations, and other issues in the NSICU patient.

The Neurovascular Axis

The relationship between certain intracranial and spinal cord structures and events impacts pulmonary vascular integrity and pulmonary and cardiac function. In addition, the mediastinal veins and pulmonary vasculature will reflect ongoing therapy aimed at addressing intracranial events. This continuum manifests on the portable chest radiograph—either directly or indirectly—as a reflection of intracranial events, through that relationship. Neurogenic pulmonary edema is a direct indication of intracranial events on the portable chest radiograph. However, pulmonary vascular engorgement or interstitial edema on the portable chest radiograph in a somewhat different but related circumstance may reflect therapeutic efforts to address intracranial pathology, and indirectly reflect its presence and status.

Neurogenic Pulmonary Edema

Fulminant pulmonary edema can develop following a variety of neurological insults. Pulmonary congestion, intraalveolar hemorrhage, and high protein edema fluid characterize this process. It is usually resistant to standard treatment measures, and, if severe, is commonly fatal (Fig. 1).[5] Neurogenic pulmonary edema (NPE) has been reported in a variety of neurological conditions, including head trauma, intracranial hemorrhage, cerebral tumors, stroke, seizures, Guillain-Barré syndrome, meningitis, poliomyelitis, venous air embolism, intrathecal hypertonic saline, and insulin shock treatment of schizophrenia (Figs. 2 and 3).[5] The precise mechanism remains unclear, but 2 hypotheses have developed from animal experiments and observations: neuronal mediation, and "blast" effect. Both alter pulmonary capillary permeability and are stimulated by increased intracranial pressure (ICP).

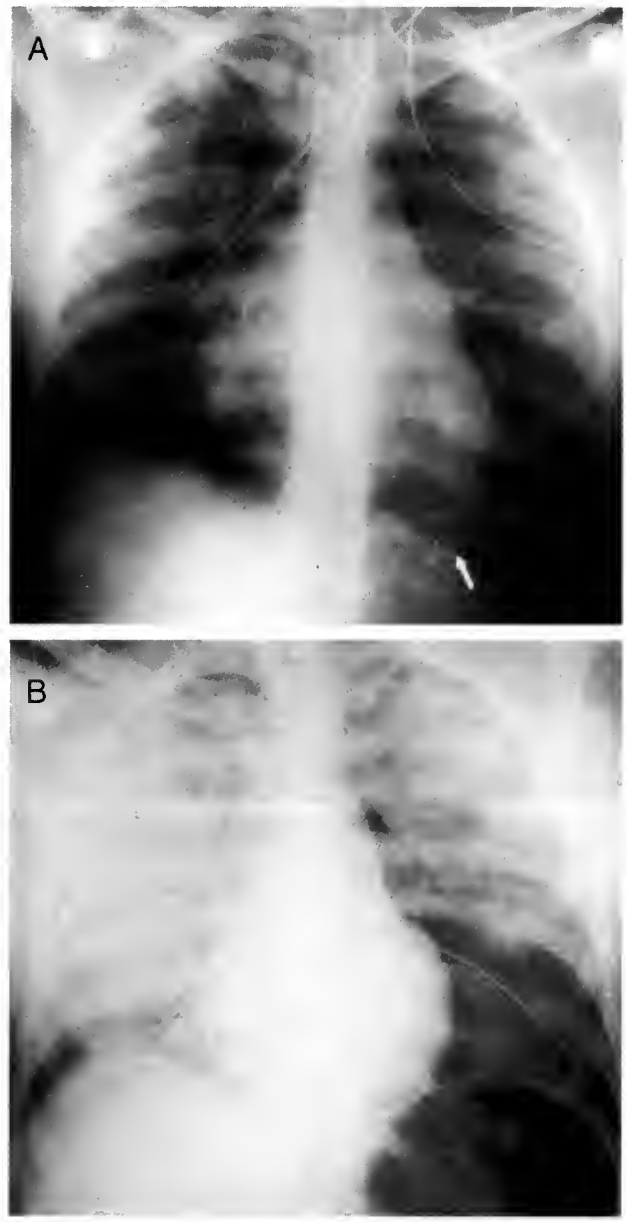

Fig. 1. A: Anteroposterior portable chest radiograph of a 30-year-old woman found unresponsive at home and brought to the emergency department. Mild interstitial edema is visible in the upper lung zones, consistent with neurogenic edema. Note the distended stomach, indicating mask ventilation and the tooth (arrow) following an attempted intubation. B: A second image, obtained shortly after Figure 1A and a seizure in the emergency department, shows florid neurogenic airspace edema.

The "blast" theory postulates that a massive sympathetic discharge produces systemic and pulmonary hypertension and increases venous return. The resulting marked increase in pulmonary blood flow and pressure produces a "blast" effect on pulmonary capillary membranes, with

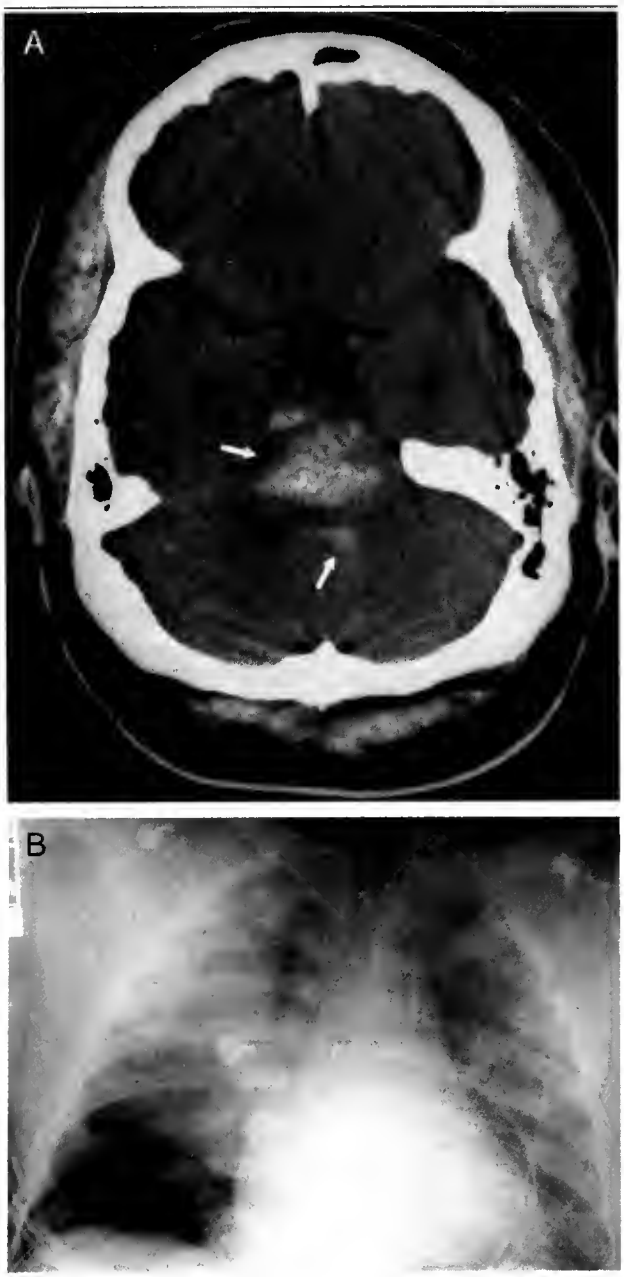

Fig. 2. A: Noncontrast computed tomography image of the brain at the level of the suprasellar cistern shows diffuse pontine hemorrhage with extension into the fourth ventricle (arrows) in a 73-year-old hypertensive patient. B: Portable chest radiograph shortly before the head computed tomography scan shows diffuse bilateral upper lobe airspace abnormality consistent with neurogenic pulmonary edema.

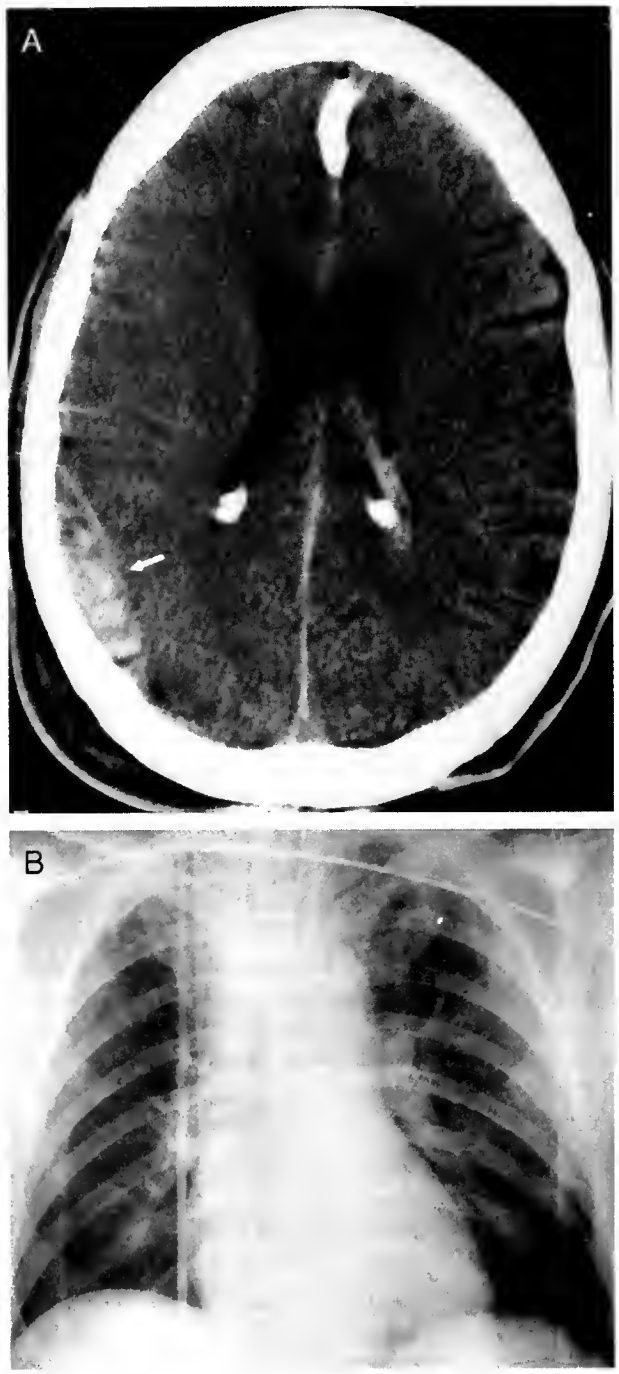

Fig. 3. A: Noncontrast computed tomography image of the brain at the level of the lateral ventricles shows a right parietal epidural hematoma (arrow) adjacent to a skull fracture seen on other images. There is also intraventricular blood, mild cerebral edema, physiologic calcifications, and soft tissue edema and irregularity at the fracture site. B: Portable chest radiograph 20 minutes before the head computed tomography shows mild vascular ill-definition consistent with neurogenic pulmonary edema in this 50-year-old male following a vehicular accident. Note multiple radiographic artifacts from a backboard.

resultant damage and, thus, capillary leak and pulmonary edema. The neuronal mediation theory supposes that stimulation of sympathetic nerves to the lung directly affects capillary permeability by altering the number and dimension of endothelial pores, thus permitting edema formation.[7] Whatever the mechanism, NPE is common in pa-

Fig. 4. Portable chest radiograph of a 59-year-old man in the emergency department after being found comatose at home following a neurological event. A mixed interstitial and airspace edema pattern is present, with a nodular component in the upper lung zones consistent with neurogenic pulmonary edema.

tients with fatal head injuries. In surviving patients, pulmonary extravascular fluid accumulation is frequent and may predispose them to increased risks of pneumonia or adult respiratory distress syndrome (ARDS).[6] Experimental evidence and clinical experience suggest that NPE can follow spinal cord injury in some patients, similar to the events following head trauma. The centrogenic pathogenesis involving intense sympathetic hyperactivity, possibly secondary to stimulation or compression of hypothalamic, medullary, or brainstem areas postulated in head injury, may also involve centers or tracts in the spinal cord.[6,7]

Radiographically, NPE presents a pattern similar to other forms of edema. It may be as subtle as interstitial edema, sometimes in a nodular pattern (Fig. 4). More commonly it occurs symmetrically as a readily-recognized airspace process. These patterns favor an upper lobe distribution in some patients, a distinction frequently mentioned in the literature and supported by my experience. The reasons for this distribution are unclear, but may relate to alterations in pulmonary venous pressure.[8] However, the majority of the time, the radiographic appearance of NPE is typical of any noncardiac type of edema, with no distinguishing features except the temporal relationship to a neurological event.

Intravascular Therapy

Intracranial hypertension (increased ICP) is thought to incite the processes leading to NPE. This elevated cranial pressure is a common finding in head injuries and other neurological events, with or without NPE. ICP reflects the contribution of 3 components: brain (80%), cerebrospinal fluid (10%), and blood (10%). Any alteration in this equation, such as the presence of a space-occupying intracranial lesion, intracranial hemorrhage, or cerebral edema, alters the relationship of these components in the rigid skull. Cerebral blood flow is a major determinant of ICP and cerebral tissue perfusion. Reduction of cerebral blood flow has highly deleterious effects on tissue perfusion. Treatment is often directed at reducing ICP in cases of intracranial hypertension and cerebral edema in order to maintain cerebral blood flow and cerebral perfusion pressure, and, thus, tissue perfusion.

Several methods are available to reduce increased ICP. One of the cornerstones of therapy for reduction of increased ICP is osmotic diuresis. Intravenous osmotic agents that do not pass the blood-brain barrier create an osmotic gradient toward the intravascular space and reduce ICP by reducing total brain water.[9] Total intravascular volume is increased, and some patients exhibit portable chest radiograph manifestations of positive fluid balance, and possibly lung edema. On the portable chest radiograph, distention of the upper mediastinal veins, prominent, enlarged pulmonary vessels, and possibly an increase in heart size suggest positive fluid balance. If the fluid balance becomes more positive, some "spill" will occur into the lungs as intravascular volume and pressure increases, resulting in interstitial or pulmonary airspace edema. A Swan-Ganz catheter is sometimes used to titrate fluid balance in difficult cases (Fig. 5).

Another therapeutic endeavor can similarly lead to signs of positive fluid balance and interstitial edema on the portable chest radiograph. In subarachnoid hemorrhage, vasospasm of cerebral vessels can produce significant neurological deficits and can be identified by arteriography in 70% of patients after subarachnoid hemorrhage. However, only approximately 30% of these patients develop delayed neurological deficits. Medical management of vasospasm includes arterial hypertension, hypervolemia, and hemodilution therapy. In this fashion, cerebral blood flow and perfusion pressures are improved and cerebrovascular resistance is decreased, thus improving circulation and oxygen delivery to ischemic cerebral tissue.[10] Hypervolemia or positive fluid balance manifests on the portable chest radiograph, and interstitial or pulmonary edema may also be seen during this therapy (Fig. 6).

These 2 therapies share a common goal: to maintain cerebral tissue perfusion. Osmotic therapy seeks to shrink edematous cerebral tissue to maintain perfusion, while hypervolemia therapy seeks to maintain perfusion by preventing vasospasm. In both cases, intravascular volume is increased and, thus, they produce similar findings on the portable chest radiograph. The temporal relationships of these therapies will likely differ, permitting distinction in some clinical situations. Reduction of cerebral edema generally occurs early in the course of treatment but is, of

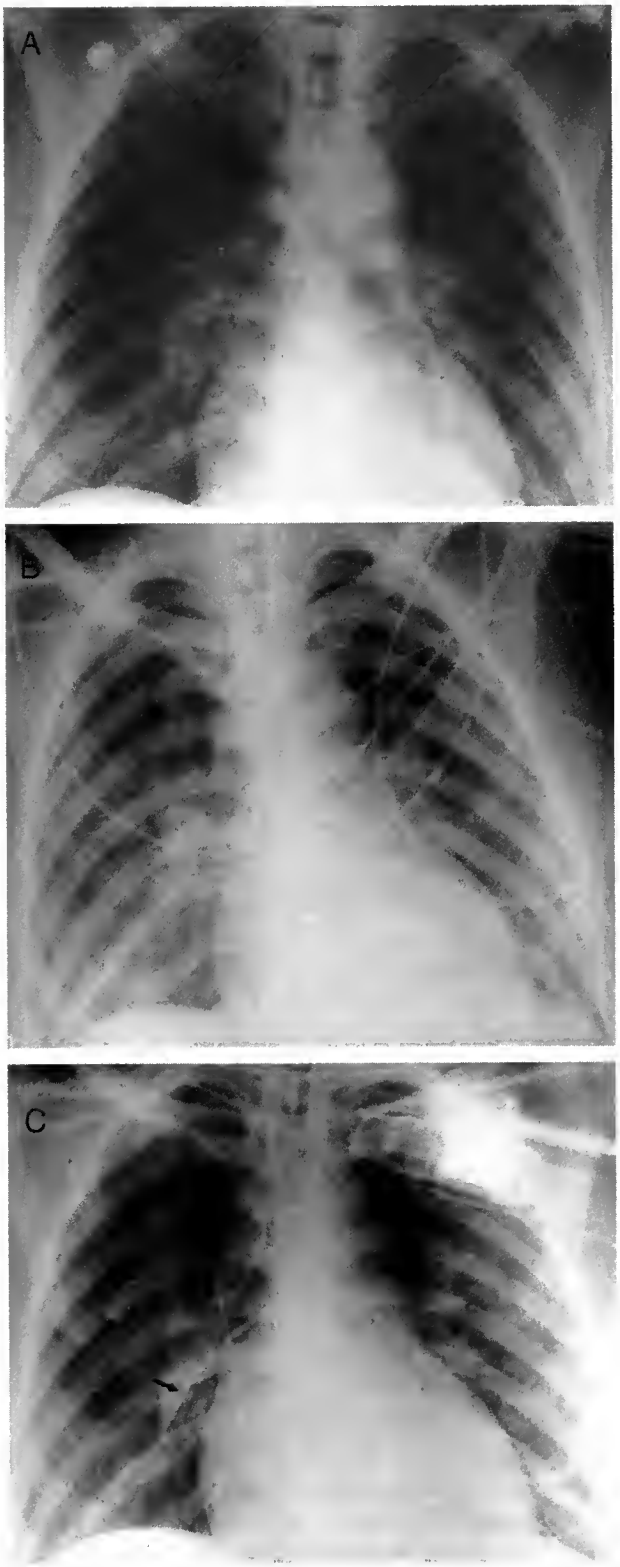

Fig. 5. A series of anteroposterior portable chest radiographs of a 54-year-old woman undergoing osmotic diuresis. A: A somewhat overexposed admission study shows clear lungs and good vascular clarity at the lung bases. B: Twenty hours later, the patient has been intubated and a large sheath catheter placed in the right subclavian vein. The upper mediastinum is wider, the hilar vessels are engorged and interstitial edema, or "spill" reduces vascular clarity. C: An additional 36 hours later, a Swan-Ganz catheter (arrow) has been inserted to allow for better fluid management. The vessels are clearer but the upper mediastinum remains widened.

course, dependent on the specific process and its severity. The treatment of vasospasm, on the other hand, generally occurs 7–10 days after subarachnoid hemorrhage, when vasospasm generally is at its maximum. Therefore, the timing of the radiographic presentation of these 2 treatments should differ, although there may be some overlap.

Thus, the neurovascular axis manifests on the portable chest radiograph, either directly as neurogenic edema or indirectly as positive fluid balance or interstitial edema in the treatment of intracranial hypertension and vasospasm. Through this relationship, the portable chest radiograph acts as a window to intracranial events. Note that in another ICU the development of interstitial edema would have an entirely different interpretation and prognosis. In a patient who had just suffered a myocardial infarct, these occurrences could be ominous, signaling myocardial injury or another complication. In the NSICU, these findings would likely indicate the need to adjust intravascular therapy in the appropriate clinical situation. This underscores the risks of misinterpretation when applying generalized rules to ICU chest radiographs without specific knowledge of the nuances of care unique to a particular ICU setting.

The Neurosciences Intensive Care Unit

The ICU environment differs somewhat, depending on the type of patient. Most of the radiographically visible appliances will be familiar, but a few will be different, and some unusual. In patients requiring chronic drainage of cerebrospinal fluid, radiographs show ventriculo-peritoneal shunts crossing the lung fields (Fig. 7). Rarely, a ventriculo-caval or ventriculo-atrial shunt will be visible. Vascular catheter placements are common, but occasionally a jugular catheter will be visible in a reverse orientation heading into the jugular system rather than entering the thoracic vessels from a jugular approach. These catheters are purposefully placed into the jugular veins and directed toward the head to sample venous blood exiting the brain for oxygen saturation or for analysis of another component to assist in determining brain metabolic activity (Fig. 8). In another care unit, that catheter positioning would be cause for concern, and replacement or repositioning would follow, but not in the NSICU. On the portable chest radiograph, several types of cervical or thoracic spine stabilizing device can appear superimposed over portions of the lungs and mediastinum, which can hinder interpretation.

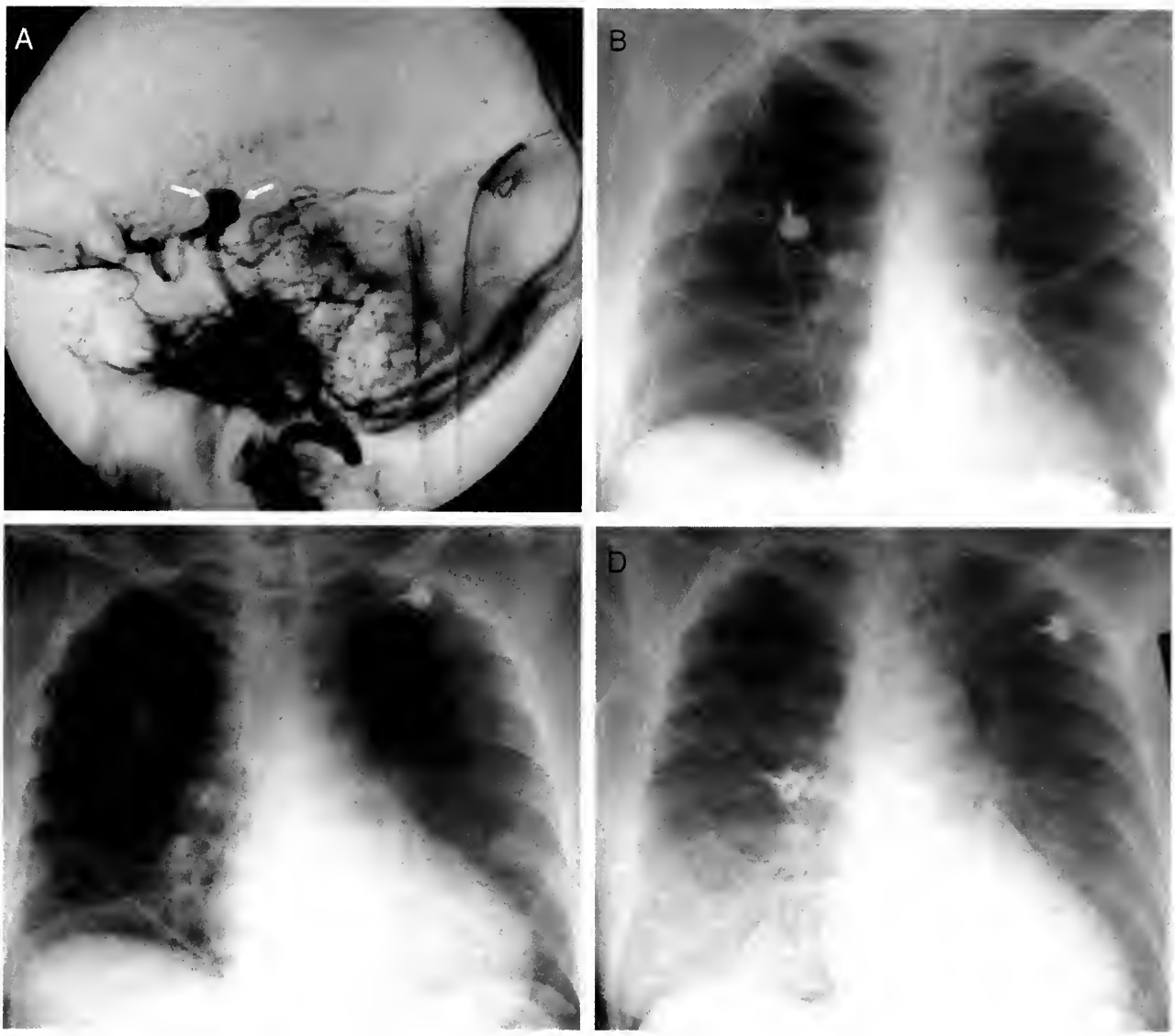

Fig. 6. A: A digitally subtracted oblique lateral view from a left vertebral artery injection shows a large basilar artery tip aneurysm (arrows) in a 61-year-old woman. Hypervolemia therapy was employed to combat vasospasm. B: Slightly rotated initial study shows endotracheal and nasogastric tubes, a large bore right subclavian sheath catheter, and clear lungs as therapy begins. C: Forty-eight hours later, the mediastinum is wider and the hilar vessels are enlarged, indicating the increased intravascular volume of hypervolemia therapy. D: Seventy-two hours into the therapy, a rotated portable chest radiograph shows interstitial and airspace edema "spill."

Immobility, a frequent clinical reality for NSICU patients, can contribute to the development of pneumonia, atelectasis, deep vein thrombosis, pulmonary emboli, pressure ulcers, urinary tract calculi and infections, muscle atrophy, contracture, and osteoporosis.[11] Manual turning is sometimes replaced with continuous rotation of the patient on a mechanically rotating bed. Continuous lateral rotational therapy with kinetic beds has been shown to reduce the length of ICU stays and the incidence of nosocomial pneumonia in treated patients.[12] These rotating beds can provide support for spinal or lower extremity traction, patient weight monitoring, and vibrating mechanisms that assist in chest physiotherapy and pulmonary toilet.

Measures intended to prevent or address the complications of immobility, utilizing continuous lateral rotational therapy on a special bed or table might be visible on the portable chest radiograph. The bed is equipped with a slot for a film cassette, which is at a fixed distance from the patient and requires that the patient fit the geometry of the fixed patient-to-film distance. Obviously, patient size will vary, and patients will not always be precisely positioned over the film cassette. Some portion of the chest might thus be excluded from the examination, and detail can be compromised somewhat by the varying geometry. In addition, some beds are designed with special hatches for access to certain body areas, and these hatches are fre-

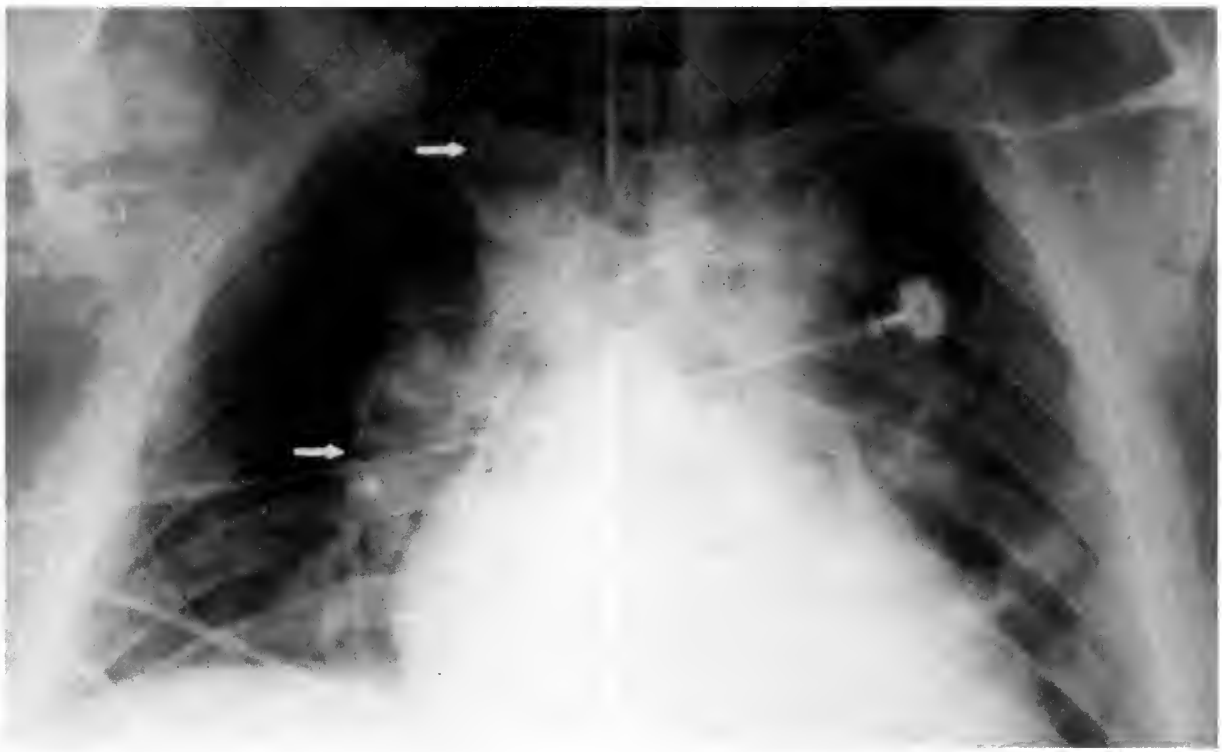

Fig. 7. Portable chest radiograph of an intubated seizure patient with a ventriculo-peritoneal shunt catheter projected across the right hemithorax (arrows). A left subclavian catheter is also present.

quently visible on the portable chest radiograph (Fig. 9). In addition to artifacts and geometrical limitations of the bed, stabilizing or traction devices for spinal injuries are sometimes projected across the thorax. Thus, the portable chest radiograph image can be a collection of tubes, lines, catheters, monitoring equipment, artifacts, and traction/stabilizing devices, intermingled with the signs of the patient's cardiopulmonary status.

Neurological Deficits Impacting Cardiorespiratory Function

A great deal of effort will obviously be directed toward addressing and managing the primary neurologic insult, be it head trauma, stroke, or cerebral aneurysm. A significant element of nonthoracic imaging will be directed at addressing the neurological insult and/or other attendant injuries or problems. The portable chest radiograph will be central to monitoring cardiopulmonary activity and detecting associated complications, and in some instances, incidental radiographic findings will reflect the neurological disorder (Fig. 10).

In terms of pulmonary or respiratory support, 3 clinical goals are uppermost: airway maintenance, adequate alveolar ventilation, and appropriate oxygenation.[13] The emphasis depends on the patient's clinical status and coexistent pulmonary conditions. Many patients will be obtunded or sedated, and most may have neurological deficits that complicate achieving the stated goals.

The endotracheal tube will be visible on the portable chest radiograph, and its tip should be 3–7 cm above the carina. This positioning allows safe excursion between the carina and the vocal cords during flexion or extension of the neck and lateral movements of the head that occur spontaneously or during suctioning.[4] Main bronchus intubation, overdistention of the endotracheal tube (ETT) cuff, and sharp angulation in the course of the tube suggesting airway injury or perforation, demand attention. The superimposition of numerous lines, tubes, and catheters with radiopaque stripes can sometimes make the identification of the ETT tip difficult. Esophageal intubation is an additional problem and, although uncommon, is difficult to detect. Oblique studies of the airway will aid in localizing the errant esophageal ETT.[14] The tracheostomy tube should be two-thirds of the tracheal width and parallel to its lateral walls. The tip is located one-half to two-thirds the distance between the stoma and the carina.[14] The stoma is difficult to accurately pinpoint.

The radiographic presence of an ETT generally implies respiratory failure and hypoxia, and the association of substantial parenchymal lung disease and the ETT is common. Intubation is sometimes seen with clear lung fields following anesthesia or in exacerbation of chronic obstructive pulmonary disease. In the NSICU, the lungs will be

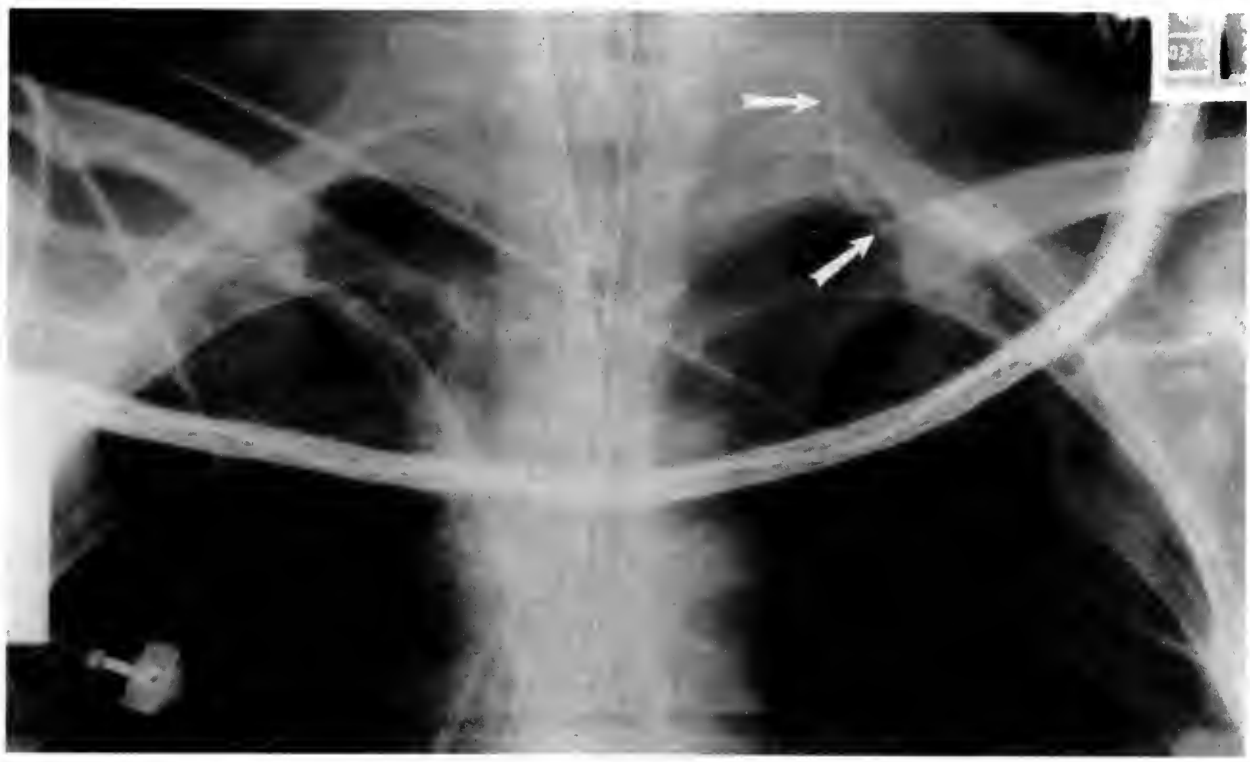

Fig. 8. Coned down view of the upper chest showing a reverse jugular catheter entering the jugular system and directed toward the head (arrows).

relatively clear, but the patient will be intubated for airway protection to prevent aspiration and in situations where central respiratory drive is lost. Hypoxia stemming from extensive lung disease is not the only indication for intubation; intubation may be performed for hyperventilation therapy for increased ICP. Hyperventilation induces hypocarbia and lowers ICP through reduction of cerebral blood volume.[9]

Lung volume can be decreased by retained secretions, minimal cough response, immobility, extravascular lung water, and shallow respirations. These problems are commonplace in the NSICU because of the additional burden of neurological impairment in these patients. Atelectasis of major proportions is common, but there seems to be a subtle distinction between patients with central cerebral insults or events and those with peripheral deficits. Patients with transected cervical spinal cords, myasthenia gravis, Guillian-Barré syndrome, amyotrophic lateral sclerosis, and other neuromuscular disorders seem to differ somewhat in the frequency and severity of atelectasis. Patients with cervical cord lesions or neuromuscular failure have significant ventilatory embarrassment due to paralysis or weakness, respectively, of the muscles of respiration. Those with higher lesions have greater mechanical respiratory dysfunction. A traumatic complete spinal cord lesion at C4 or above, if tolerated, would result in the scalene, trapezius, and sternocleidomastoid muscles being the only respiratory muscles. Below C4, diaphragmatic and cervical accessory muscles are maintained. Intercostal muscles supplied by T1-T12 and abdominal muscles innervated by T7-L1 are paralyzed. Respiratory compromise stems from both inspiratory and expiratory muscle weaknesses. The rib cage is not stabilized, lung volumes are diminished, and adequate cough is lost.[1] These compromises of respiratory muscle function lead to an inability to clear the airways, resulting in more common and frequent lobar collapse (Fig. 11). Patients with polyneuropathies may suffer weaknesses of the bulbar musculature, resulting in loss of upper airway protection, inability to swallow, and risk of aspiration. In addition, chest injuries accompanying head or spinal injuries in trauma patients may predispose to a greater frequency of aeration disturbances. These patients are also at increased risk of aspiration and pneumonia.

Aspiration can be seen following head trauma and stroke. It generally occurs prior to hospitalization, sometimes as a complication of the injury, or of intubation. Seizure patients sometimes aspirate during attempts to force utensils into their mouths to "protect" them. Aspiration does occur in the NSICU, although staff vigilance and preventive measures are poised to minimize its frequency. Aspiration can occur around a high-volume, low-pressure ETT, although it is not common, and particulate matter is absent.[13] It is important to document aspiration, preferably by the obser-

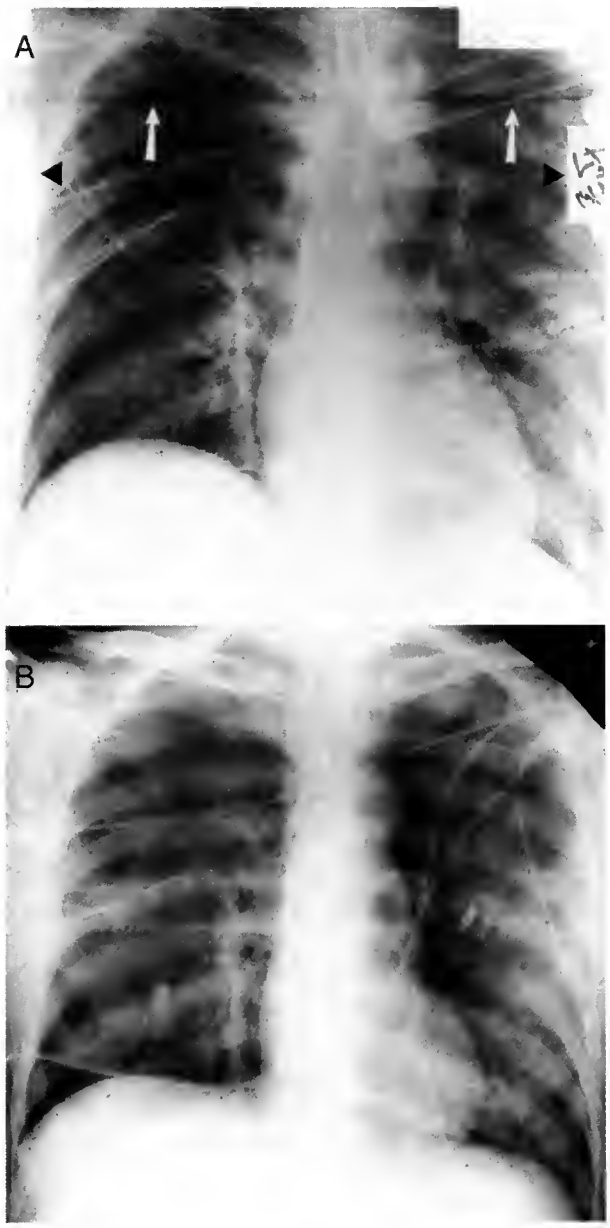

Fig. 9. A: Portable chest radiograph of a 27-year-old man on a rotating bed following head trauma. Note the right chest tube, right rib fractures, tracheostomy tube, slight vascular haziness, and that a portion of the left chest is excluded. The rectangular shadows at the periphery (arrowheads) and the horizontal lines from one of the bed flaps (arrows) are related to the kinetic bed. B: Portable chest radiograph of the same patient on the same day on a standard bed. Note the magnification of structures in Fig. 9A compared to 9B. Detail, especially vascular clarity, is improved in Fig. 9B, as is patient positioning.

vation of a health care provider. Not every deviation from clinical baseline implies aspiration, as it sometimes seems. Other clinical occurrences are also possible, such as arrhythmia, extension of the neurological process, or pulmonary embolus.

Radiographically, aspiration generally presents as an airspace process in a dependent portion of the lung, and is usually a relatively focal abnormality, unless a massive aspiration has occurred. The posterior segments of the upper lobes and the superior and posterior basilar segments of the lower lobes are more frequently involved. The appearance will vary somewhat depending on the characteristics of the aspirate. Relatively benign material such as saliva or blood will usually present as a loosely consolidative, pneumonic type opacity that resolves relatively quickly. Aspirates with endobronchial particulate matter may add volume loss to the radiographic picture. Low pH material will act as a pulmonary "burn," stimulating an edematous response in the affected area as the involved lung attempts to neutralize the offending agent.

In some cases of aspiration, a small nodular or acinar pattern can be seen at the periphery of the pulmonary opacities and may suggest the diagnosis (Fig. 12). This acinar pattern is also sometimes seen in pulmonary hemorrhage, especially following intubation and ventilatory support.

Neurogenic pulmonary edema does not always occur as a transient, rapidly resolving, self-limited process in the emergency department prior to admission to the NSICU. It can persist or present as a malignant process with severe hypoxia and copious, foamy secretions.[1] Therapy directed at the primary neurologic problem may have to be modified or delayed until this process is brought under control. Intubation, vigorous pulmonary toilet, administration of positive end-expiratory pressure, high inspired oxygen, and diuresis may be necessary.[1] Some authors have reported 2 forms of NPE: acutely, within minutes to hours, or, more insidiously, occurring over a period of days following central nervous system insult.[6]

Sequelae of Care

NSICU patients experience a variety of therapies and events during their hospital course, and the critical care setting itself can contribute to complications. The presence of tubes, catheters, and other invasive appliances compromise natural defense mechanisms and can put the patient at risk for infection. Prolonged recumbency and immobility in the course of therapy for increased ICP, skeletal trauma, or as a sequela of the neurologic insult can predispose the patient to thromboembolism. Patients suffering trauma may have injuries that require chest tubes, splints, or other support or therapeutic devices. Thus, the clinical picture can become very complicated, and several ongoing clinical problems can manifest on the portable chest radiograph.

Nosocomial pneumonia is the most common infectious problem in this patient population, and is a direct contrib-

Fig. 10. A: Portable chest radiograph of a 27-year-old patient following a seizure. Note the bilateral shoulder dislocations (asterisks) suffered during the seizure, and the enlarged heart. B: Post-reduction with humeral heads normally positioned.

utor to morbidity. Depressed consciousness, intubation and ventilatory support, changes in oropharyngeal and gastric bacterial flora, and exposure to health care personnel have been shown to be instrumental in the development of pulmonary infections. In addition to intubation, aspiration, inadequate pulmonary toilet, generalized debilitation, and inappropriate use of broad spectrum antibiotics are all additional factors. Establishing the diagnosis can be difficult but is critical, because fever can derive from many sources and can raise ICP. Portable chest radiography is not a specific tool to distinguish noninfectious from infectious processes. The Centers for Disease Control have published guidelines for the diagnosis of nosocomial pneumonia for epidemiological studies.[15] In addition to purulent sputum or evidence of an organism, physical findings of pneumonia or infiltrate on the portable chest radiograph must be present $>$ 72 hours after admission. Therefore, evaluating the sequence of findings since admission on the portable

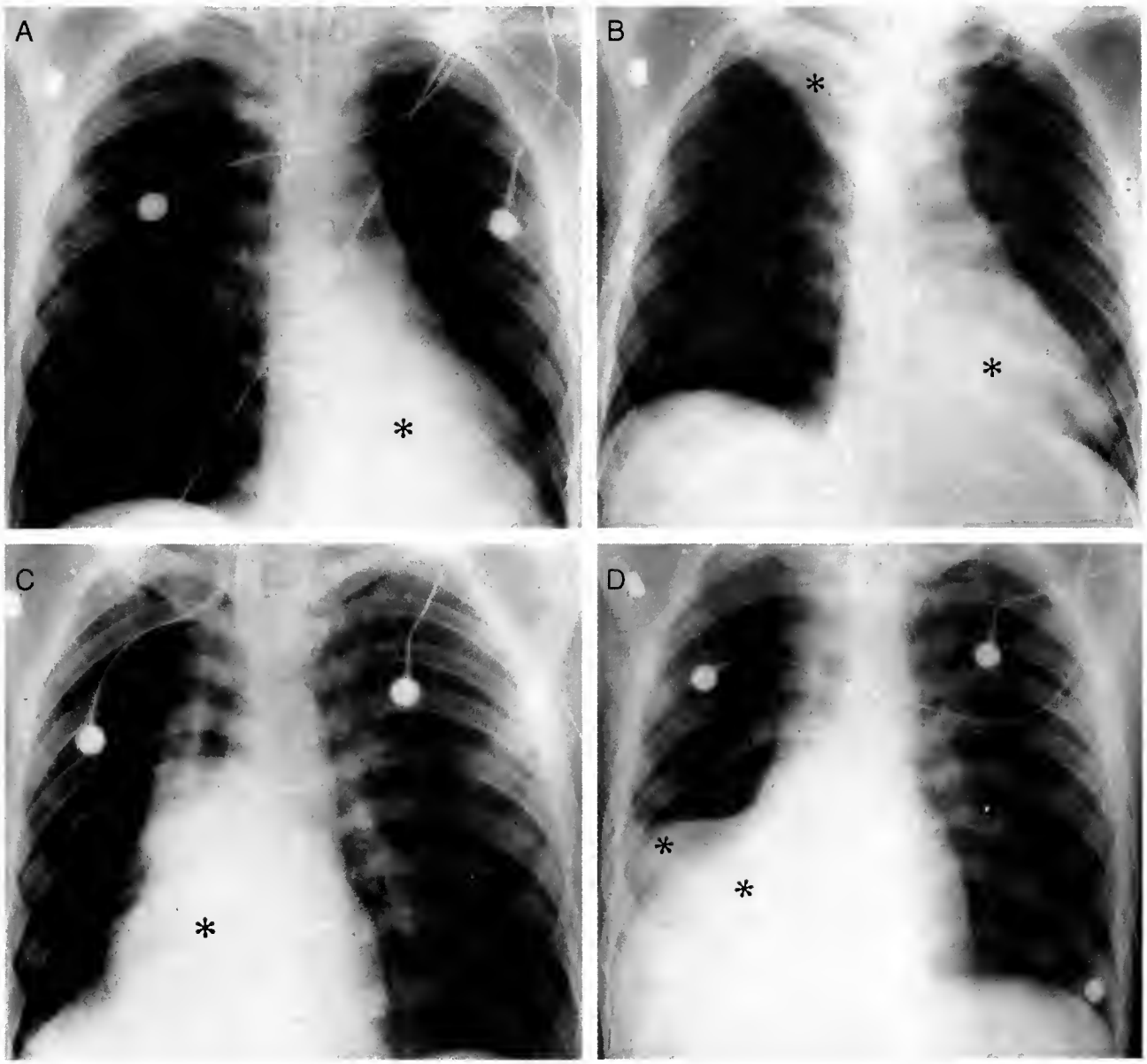

Fig. 11. A series of portable chest radiographs over a brief period of a 26-year-old man following gunshot injury transecting the cord at C7. Rotational lobar collapse is demonstrated. Note the bullet fragments in the right axilla. A: Collapsed and airless retrocardiac left lower lobe (asterisk). B: Collapsed right upper and left lower lobes (asterisks). C: Re-expanded right upper and left lower lobes. Newly collapsed retrocardiac right lower lobe (asterisk). D: Combined collapse of right middle and lower lobes (asterisks). (From Reference 4, with permission.)

chest radiograph, and other clinical data in the proper time frame, will assist diagnosis (Fig. 13).

ARDS is seen in the NSICU in situations of sepsis and generalized, multisystem trauma. Radiographic signs of ARDS may be seen soon after admission, but more commonly develop over a period of a few days. Initially ARDS will appear similar to hydrostatic edema, but the more widespread distribution of the airspace process, its resistance to diuresis, and hypoxia will suggest the diagnosis of ARDS. Positive pressure ventilation, if required for treatment of ARDS, may complicate the treatment of increased ICP by elevating intrathoracic pressure and inhibiting venous return from the head.

Reduction and control of increased ICP require immobilization and recumbency. Head injury coupled with extensive skeletal injuries usually requires protracted immobilization and recumbency, which causes significant risk of deep vein thrombosis and pulmonary embolism. Thrombi frequently develop in the veins of the thigh and pelvis. Preventive measures such as external pneumatic compres-

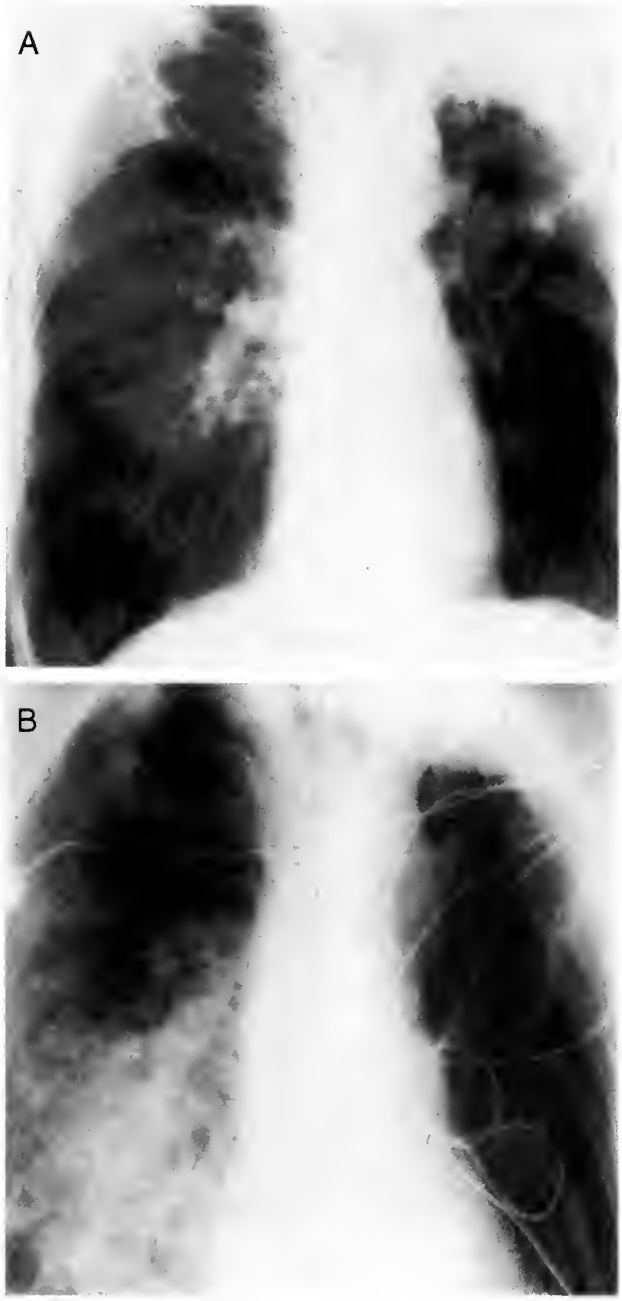

Fig. 12. A: Admission chest radiograph on an elderly male with symptoms of stroke. There is evidence of emphysema, biapical scarring, and left hilar retraction from previous tuberculosis. B: Portable chest radiograph shortly after admission and a witnessed aspiration shows a right lower lobe airspace process. Note the small nodular character of the pneumonia at the lateral and superior edges of the process.

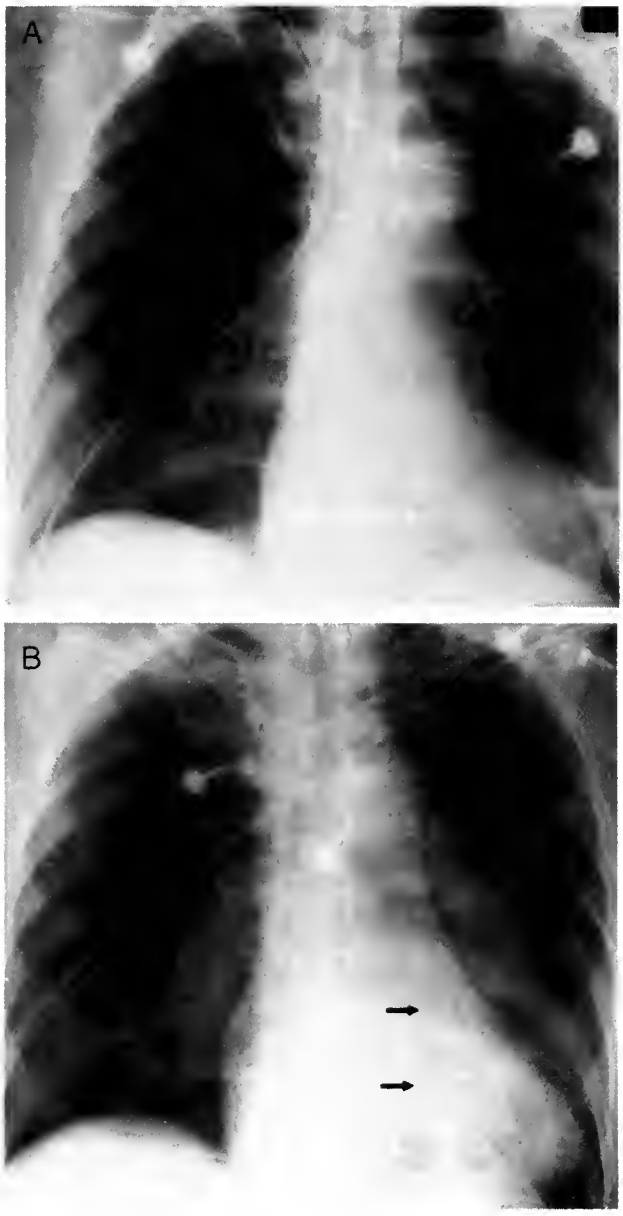

Fig. 13. A: Intubated 66-year-old male with Guillian-Barré syndrome suffering from respiratory failure. The radiograph shows clear lungs except for linear-band atelectasis at the left base. B: Seventy-two hours later the patient, still intubated, develops fever and a new left lower lobe airspace process with air bronchograms (arrows) consistent with pneumonia. Gram-negative organisms were isolated.

sion and elastic stockings are commonly employed. Anticoagulation is problematic in recently traumatized patients and stroke patients. In high-risk patients, especially those patients immobilized for a considerable period with skeletal injuries, an inferior vena cava filter is employed. In spite of preventive measures and portable ultrasound evaluation of the venous structures, diagnosis is difficult, as it is in many other patient populations. Movement can cause embolization of vascular thrombi (Fig. 14).

Fat embolism is an additional consideration in the context of hypoxia; it may occur in head-injured patients with skeletal injuries, particularly the pelvis and long bones. Fat droplets enter the blood stream from disrupted bone mar-

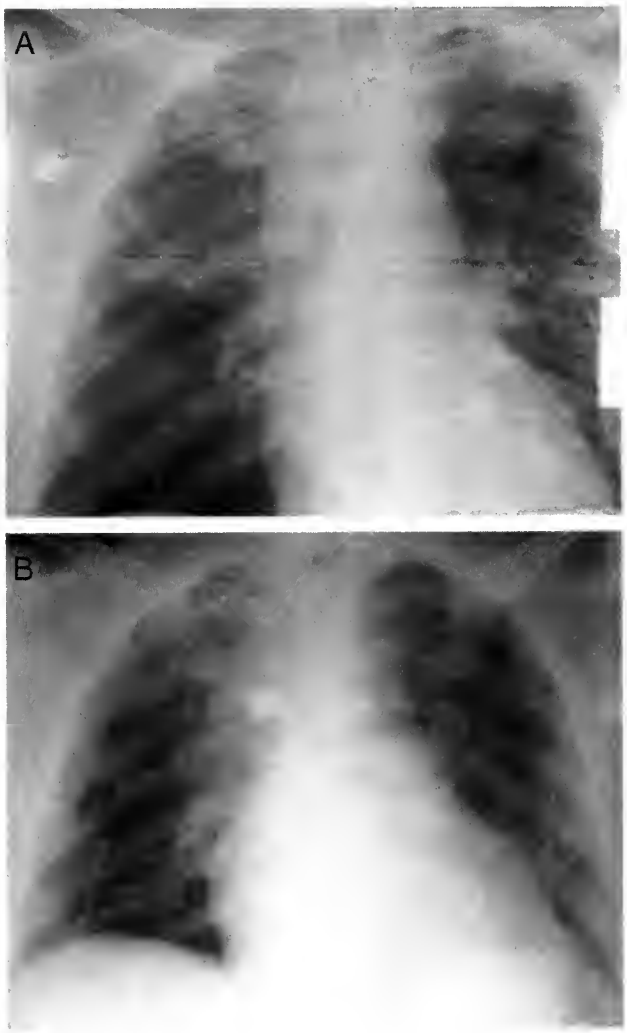

Fig. 14. A: Portable chest radiograph of a 35-year-old man on a rotating bed following severe head trauma. A portion of the left chest is excluded. Endotracheal and nasogastric tubes and a right subclavian catheter are present. Linear atelectasis is seen in the right upper lobe. Opacity in the left perihilar area relates to a pulmonary contusion. Seven days later, the patient suffered a cardiopulmonary arrest while being moved to a stretcher for a computed tomography scan. B: A portable chest radiograph obtained on the stretcher during unsuccessful resuscitative efforts. Note the enlarged right hilum in contrast to Fig. 14A, reflecting the embolic burden verified at autopsy. There is also a subtle decrease in blood flow to the right upper lobe.

row and soft tissues. The fat droplets embolize to the lungs and undergo a degradation process that damages the pulmonary capillary endothelium, leading to pulmonary edema. Fat embolism generally occurs 12–72 hours after the skeletal injury and can be accompanied by involvement of other organs, including the brain, kidneys, and skin. Radiographically, a pattern of pulmonary edema is seen in the appropriate time frame in moderate to severe cases, while milder episodes produce no radiographic finding.[16]

Fig. 15. Portable chest radiograph of the same patient as in Fig. 6. Postoperative, sternal sutures are evident following median sternotomy and repair of the aneurysm. These findings are more common following cardiac surgery. In this instance, deep hypothermia was elected for the aneurysmal repair, and cardiopulmonary bypass was performed through the median sternotomy.

Fig. 16. A postoperative portable chest radiograph of a 36-year-old man following insertion of stabilizing rods and struts in the surgical approach to a thoracic syrinx. Portions of the left third and fourth ribs have been removed, and the left hemithorax has been entered, necessitating the chest tubes. Note the deformity of the left humeral head (arrow), suggestive of a neuropathic joint (a reflection of the loss of pain and temperature sensation in this disorder).

Postoperative Situations and Attendant Issues

Following neurosurgical procedures, postoperative care occurs in the NSICU. The vast majority of these patients have portable chest radiographs on a daily basis until extubated or stable and discharged from the ICU. The radio-

graphic issues are generally the same as for nonthoracic surgery patients: appliances, lung aeration, fluid balance, lung water, fluid or air collections, and complications such as pneumonia or pulmonary embolus. In post-neurosurgery patients, advanced age, tumor type (glioma and meningioma), cardiac insufficiency, preoperative disturbances of consciousness, and preoperative corticosteroid treatment are risk factors for postoperative pneumonia. In addition to immobility and limb weakness, advanced age and malignancy in the neurosurgery patient are contributing factors for venous thrombosis and pulmonary embolism.[17]

In some cases of spinal surgery, the thorax is entered, and the evaluation must then include this consideration and its additional risks and complications. Some postoperative presentations are perplexing for the NSICU, because measures and appliances employed for other types of surgery are occasionally seen (Fig. 15). In addition, some correlated findings will occur away from the site of surgery (Fig. 16).

Summary

The evolution of intensive care into highly specialized units of subspecialty care has forced reconsideration of generic approaches to the interpretation of portable chest radiographs. Not only will appliances differ, but unique treatment methods will produce findings that can easily be misinterpreted. In order to avoid this problem, familiarity with the specific clinical entities, the approach to care by the sub-specialists, and the current treatment protocols in particular ICUs or institutions will be of great assistance.

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Chest Imaging in the Surgical Intensive Care Unit

Jeffrey S Klein MD

Introduction
Detection of Complications Following Thoracotomy
Post-Pneumonectomy
Post-Lobectomy
Post-Esophagectomy
Role of Computed Tomography in the Surgical Intensive Care Unit Patient
Pneumothorax
Pleural Effusion/Empyema
Mediastinal Collections
Image-Guided Thoracic Interventions in the Surgical Intensive Care Unit
Lung/Mediastinal Biopsy
Thoracentesis/Pleural Drainage
Mediastinal Collections
[Respir Care 1999;44(9):1078–1094] *Key words: thoracic surgery, postoperative complications, portable radiography, computed tomography.*

Introduction

Radiology in the surgical intensive care unit (SICU) provides a reliable means of monitoring the postoperative patient and detecting complications of surgical procedures. This review addresses three topics relating to postsurgical imaging: detection of complications following thoracotomy; the role of CT in imaging postoperative patients with sepsis or respiratory failure; and the use of image-guided thoracic interventional procedures in the SICU patient.

Detection of Complications Following Thoracotomy

The recognition of serious complications following thoracotomy requires a knowledge of the normal post-thoracotomy changes seen on chest radiographs.

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Post-Pneumonectomy

Immediately after pneumonectomy, radiographs typically show air in the post-pneumonectomy space, with the mediastinum in midline position (Fig. 1A). Over the first few postoperative days, the mediastinum shifts toward the operated side as air is resorbed and fluid accumulates in the post-pneumonectomy space (Fig. 1B). Complete opacification of the operated side takes 4–6 weeks.

The most common early ICU complication of pneumonectomy is pulmonary edema in the remaining lung.[1] Most cases develop within 48 hours of pneumonectomy and require mechanical ventilation for respiratory support.

Bronchopleural fistulae are more common following pneumonectomy (3%) than following lobectomy or segmental resection.[2] Bronchopleural fistulae are usually detected on upright postoperative radiographs as a falling air/fluid level in the post-pneumonectomy space, sometimes accompanied by contralateral mediastinal shift. Most bronchopleural fistulae in the early (2–3 weeks) postoperative period are the result of bronchial stump dehiscence (Fig. 1C), while delayed bronchopleural fistulae are often the result of postoperative empyema.

Cardiac torsion is a rare complication of pulmonary resection, and most often follows intrapericardial pneumonectomy.[3] This complication is recognized by a shift in cardiac position toward the side of resection, and is clinically associated with shock.

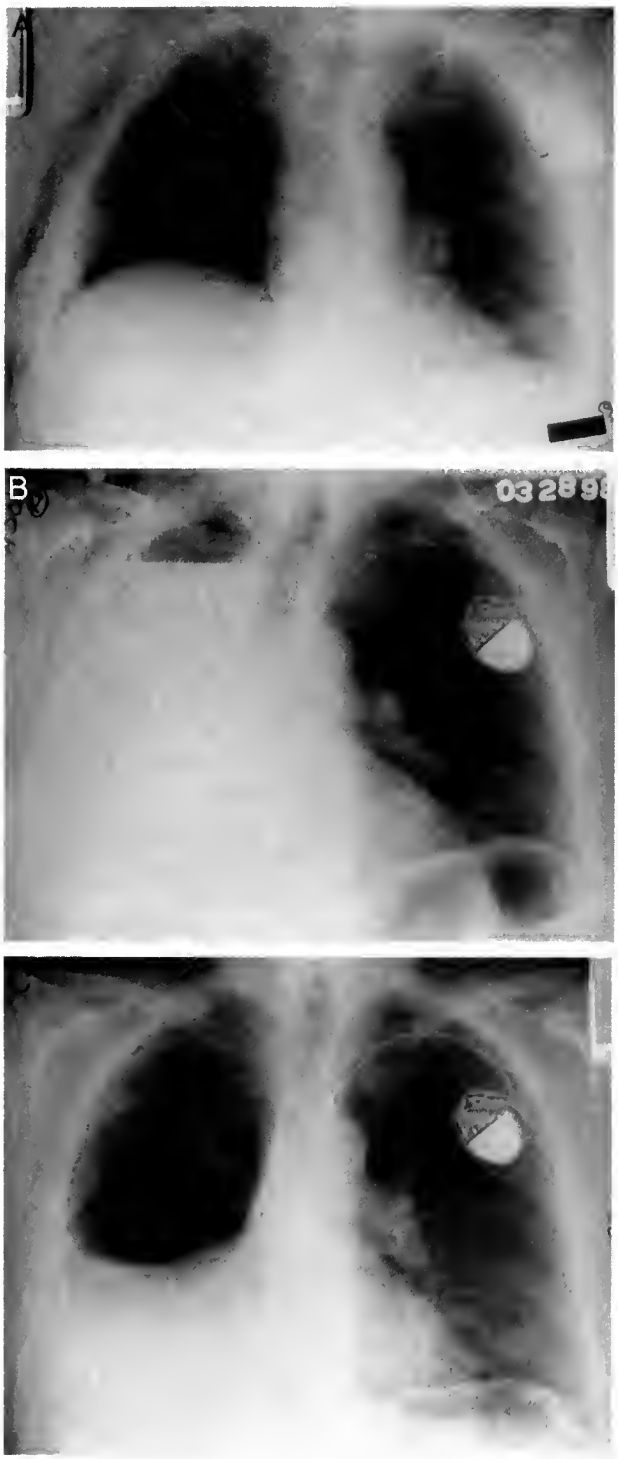


Fig. 1. Post-pneumonectomy bronchopleural fistula. A: Immediate postoperative radiograph of a patient who underwent pneumonectomy for bronchogenic carcinoma shows air in the right post-pneumonectomy space, with the mediastinum positioned midline. B: Image obtained on the second postoperative day shows fluid accumulating in the right hemithorax, with an apical air fluid level. Note the shift of the mediastinum toward the operated side. C: Two weeks after pneumonectomy, there is more air in the post-pneumonectomy space, with return of the mediastinum toward the midline. Bronchoscopy confirmed a leak at the main bronchial stump.

Post-Lobectomy

Radiographic changes following uncomplicated lobectomy are generally unremarkable, with findings including subsegmental atelectasis and small pleural effusions. Mediastinal shift and ipsilateral diaphragm elevation are common. Occasionally, fluid will rapidly accumulate in the post-lobectomy space and simulate lobar atelectasis. A small pneumothorax may be seen, and is usually the result of a temporary air leak from the surface of the remaining lung, stapled to close a fissure and facilitate lobectomy.

Complications following lobectomy are uncommon. Pulmonary complications occur in approximately one-third of patients, and include atelectasis (sometimes requiring bronchoscopy), pneumonia, edema (Fig. 2), and pulmonary embolism. Bronchopleural fistulae and empyema are less common in this group of postoperative patients.

Lung torsion is a rare and often fatal complication that most often follows release of the inferior pulmonary ligament for lung resection.[4] Rapid opacification of a lobe or lung, particularly if associated with an unusual hilar configuration or an unusual shape to the lobe, should suggest this diagnosis.

Post-Esophagectomy

Most esophageal resections in adults are performed for esophageal carcinoma. Post-esophagectomy complications include leaks from dehiscence at the esophagogastric anastomosis, empyema, lung abscess, and (rarely) chylothorax from disruption of the thoracic duct. Water soluble contrast ingestion and CT are often employed in the post-esophagectomy patient to evaluate for these complications (Fig. 3).

Role of Computed Tomography in the Surgical Intensive Care Unit Patient

While there are logistical limitations in obtaining CT studies in the SICU patient, CT is clearly more accurate than portable bedside chest radiography in the detection and characterization of postoperative intrathoracic air and fluid collections.

Pneumothorax

Patients with persistent, loculated, or unsuspected pneumothorax producing respiratory compromise, or those inadequately treated by existing chest tubes are best evaluated with CT, with drainage catheters placed at the time of the scan (Figs. 4 and 5).[5]

Pleural Effusion/Empyema

The ability of supine or semi-erect portable chest radiographs to detect even large pleural fluid collections is sig-

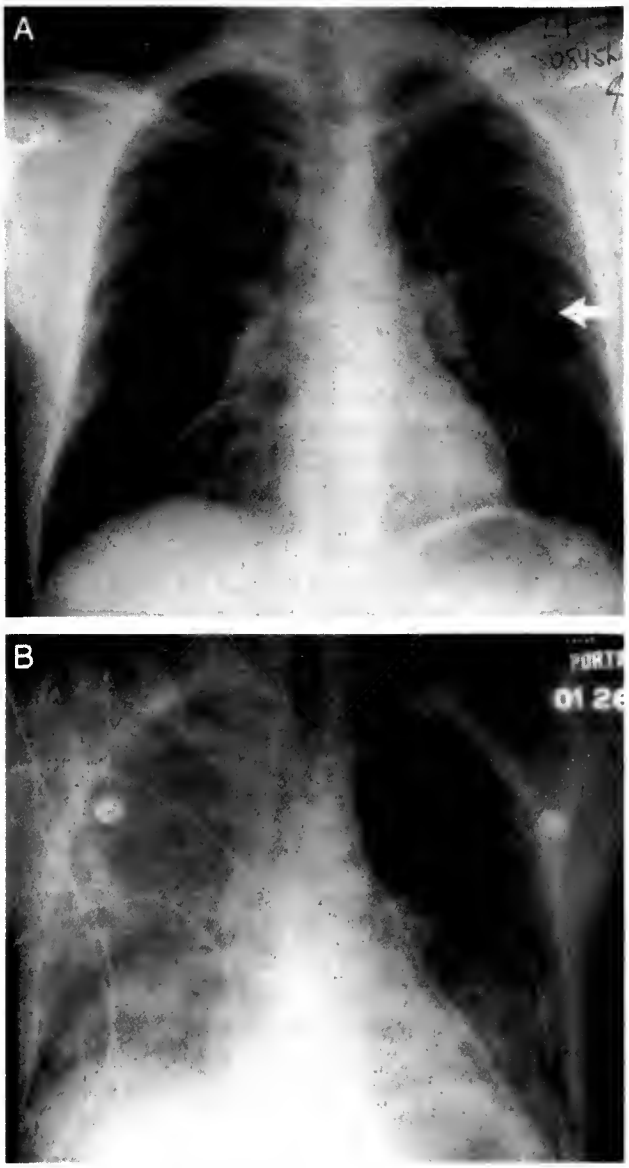


Fig. 2. Post-lobectomy pulmonary edema. A: Frontal radiograph shows a nodule (arrow) in the left mid-lung; transthoracic biopsy indicated bronchogenic carcinoma. B: Image obtained 6 hours postoperatively shows asymmetric airspace opacification representing pulmonary edema. The opacities resolved spontaneously within 5 days.

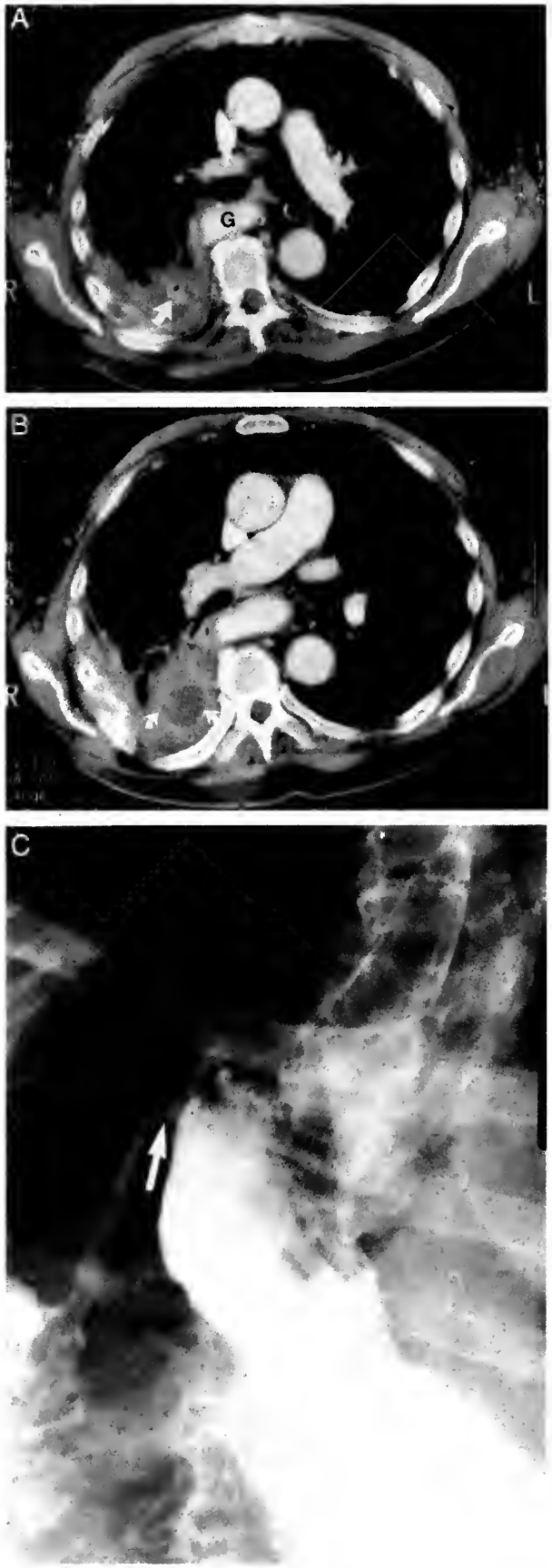


Fig. 3. Post-esophagectomy anastomotic leak. A: Computed tomography image at the level of the right upper lobe bronchus after administration of both intravenous and oral contrast shows extraluminal contrast within the right posterior chest (short straight arrow). G = gastric pull-through. B: Scan 2 cm inferior to Fig. 3A shows a fluid collection with peripheral enhancement in the superior segment of the right lower lobe (curved arrows), representing a lung abscess. C: Esophagram performed with water-soluble contrast confirms contrast extravasation (long straight arrow) at the level of the esophagogastric anastomosis.

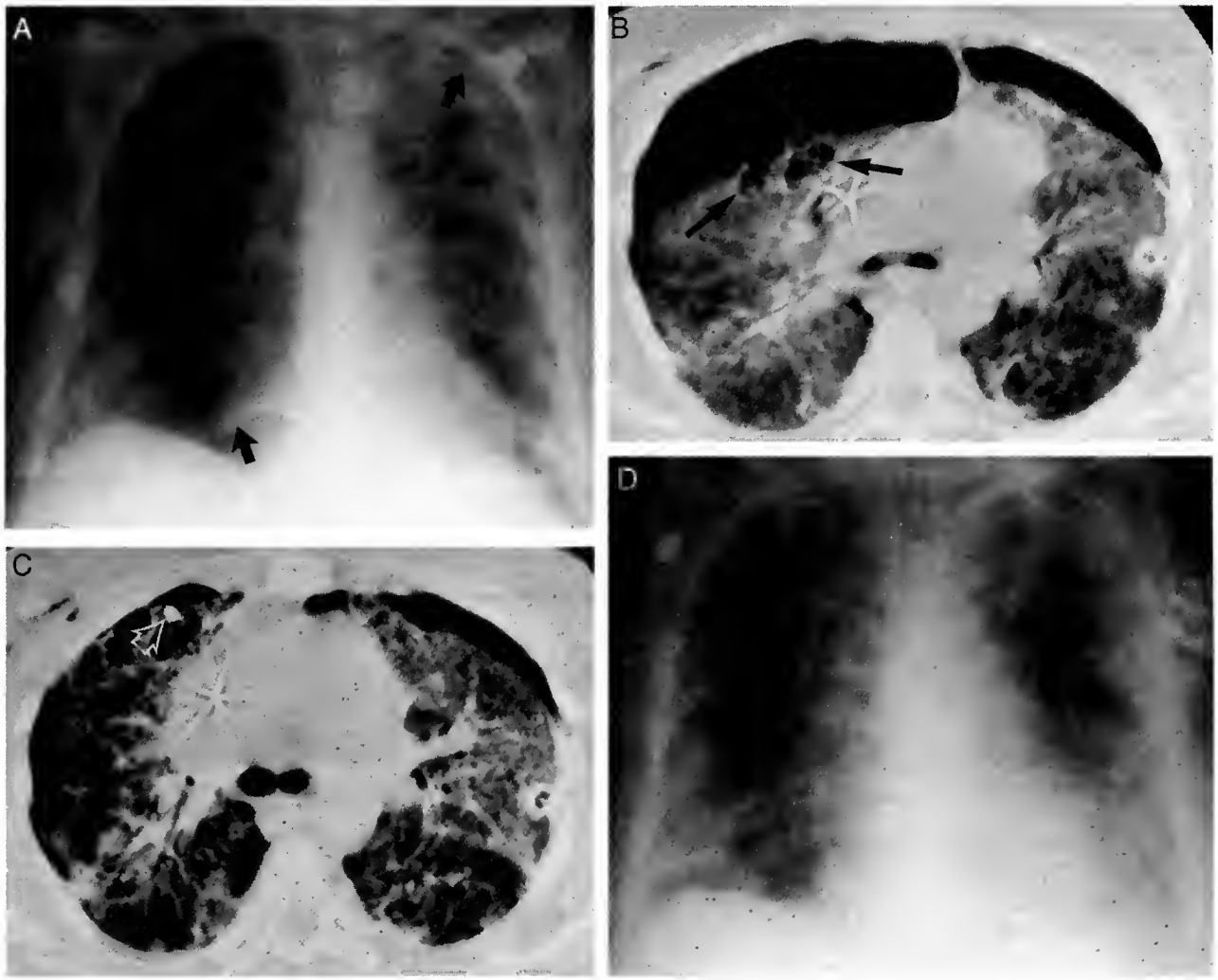


Fig. 4. Computed tomography (CT) detection and drainage of pneumothorax. A: Portable chest radiograph of a patient with acute respiratory distress syndrome and worsening respiratory status shows bilateral interstitial opacities consistent with diffuse alveolar damage. Note the presence of bilateral chest tubes (short straight arrows) placed for barotrauma-induced pneumothoraces. B: CT scan shows bilateral pneumothoraces. Note the subpleural blebs (long straight arrows) within the anterior segment of the right upper lobe that probably account for the air leak and pneumothorax. C: Repeat CT after percutaneous placement of a right pleural drainage catheter shows the catheter (open arrow) within the anterior pleural space, and evacuation of the pneumothorax. D: Repeat chest radiograph shows the pigtail catheter with its tip in the right apex. The patient eventually recovered.

nificantly limited. Although decubitus radiographs can be obtained in the SICU setting, they are technically difficult to obtain and fail to detect loculated collections. CT has proven useful in detecting and quantifying pleural collections in the postoperative patient. In cases of suspected postoperative empyema, CT-guided percutaneous drainage provides a safe and effective alternative to surgical thoracostomy or thoracotomy (Fig. 6).

Mediastinal Collections

Post-thoracotomy mediastinal collections most often represent mediastinal hematomas or seromas and require no therapy. In the post-esophagectomy patient with a mediastinal fluid collection and fever or failure to thrive, the differential diagnoses include abscess formation and chylous fluid collection (Fig. 7), the latter resulting from disruption of the thoracic duct following mediastinal dissection. CT-guided aspiration and drainage can be useful in these patients.

Image-Guided Thoracic Interventions in the Surgical Intensive Care Unit

Postsurgical percutaneous drainage of intrathoracic fluid collections can temporize a patient who requires reexploration, or may serve as the definitive drainage procedure.

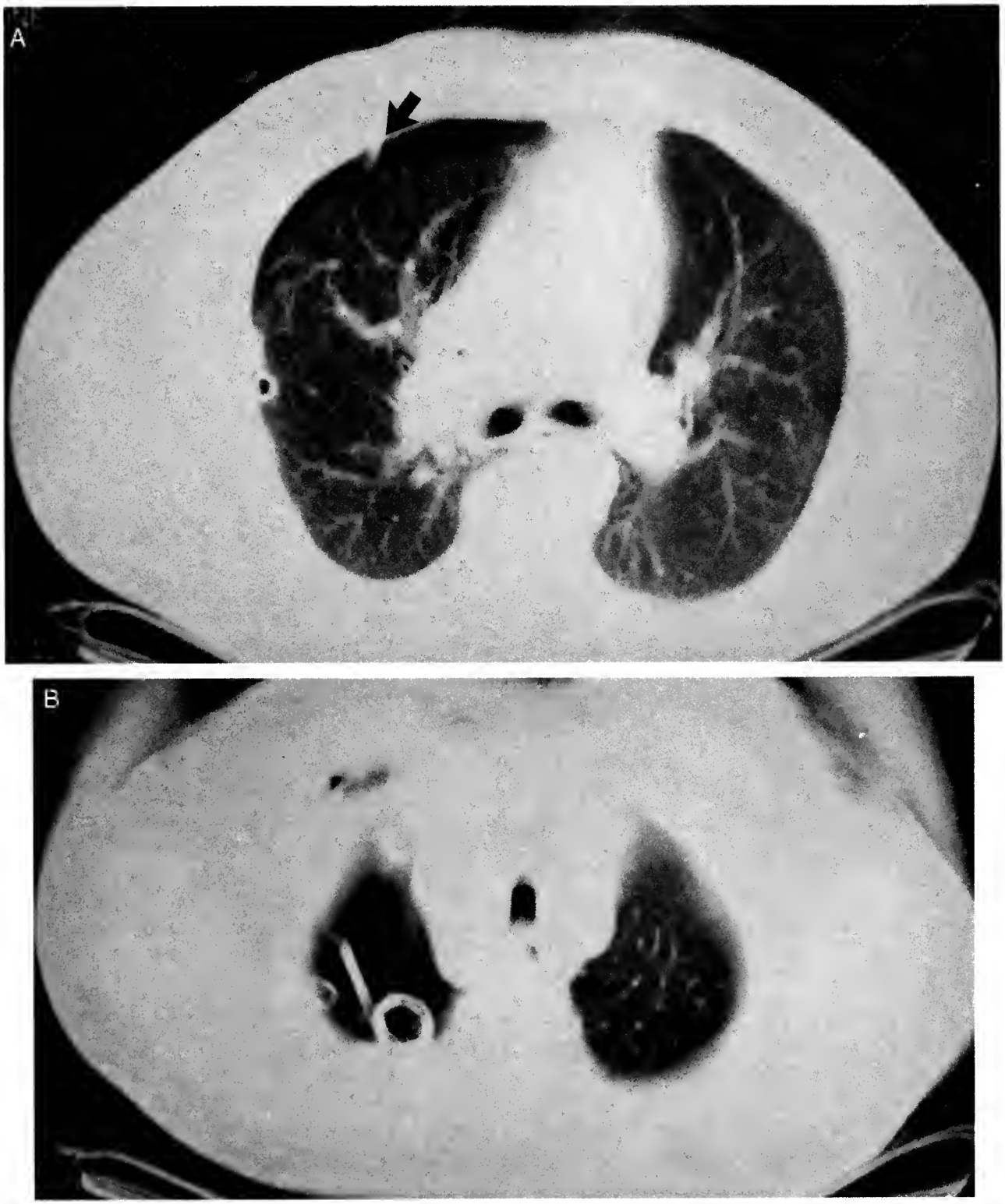


Fig. 5. Computed-tomography (CT)-guided drainage of post-lobectomy pneumothorax. A and B: CT scans obtained through the upper thorax after placement of a pleural drainage catheter for a persistent pneumothorax following right upper lobectomy shows the catheter entering anteriorly (arrow in A), with the tip at the pleural apex. C: CT anteroposterior scout view shows the drainage catheter in position, with a persistent apical pneumothorax. D and E: Repeat CT scans after 5 minutes of suction applied to the drainage device shows evacuation of the pneumothorax, with reexpansion of the lung. (*Figure continues on next page.*)

Fig. 5. *Continued.* (*Figure continues on next page.*)

Fig. 5. *Continued*

Computed tomography and ultrasound are employed for the majority of these procedures.

Lung/Mediastinal Biopsy

On rare occasions, a transthoracic needle biopsy of a lung or mediastinal mass may be requested in a SICU patient to diagnose an intrathoracic mass or nodule. This is invariably performed under CT guidance, with specimens obtained for both cytopathology and microbial stains and cultures (Fig. 8).

Thoracentesis/Pleural Drainage

Bedside ultrasound-guided thoracentesis is a safe diagnostic technique in the SICU patient, particularly those on mechanical ventilation. Loculated collections and empyemas are best approached using CT guidance with pigtail catheters attached to underwater suction devices.[6] Intrapleural lytic therapy using urokinase is effective in Stage I and Stage II parapneumonic effusions, and can obviate more aggressive surgical management of these postoperative collections (Fig. 9).[7]

Pneumothorax that is either loculated or remote from indwelling thoracostomy tubes is easily managed by CT-guided catheter drainage, with a success rate exceeding 90%.

Mediastinal Collections

Infected mediastinal fluid collections most often complicate median sternotomy, and require surgical drainage because of the high mortality associated with these collections. CT can be used to detect and aspirate infected mediastinal collections preoperatively.[8] Noninfected mediastinal collections that may require percutaneous drainage include seromas and chylous collections.

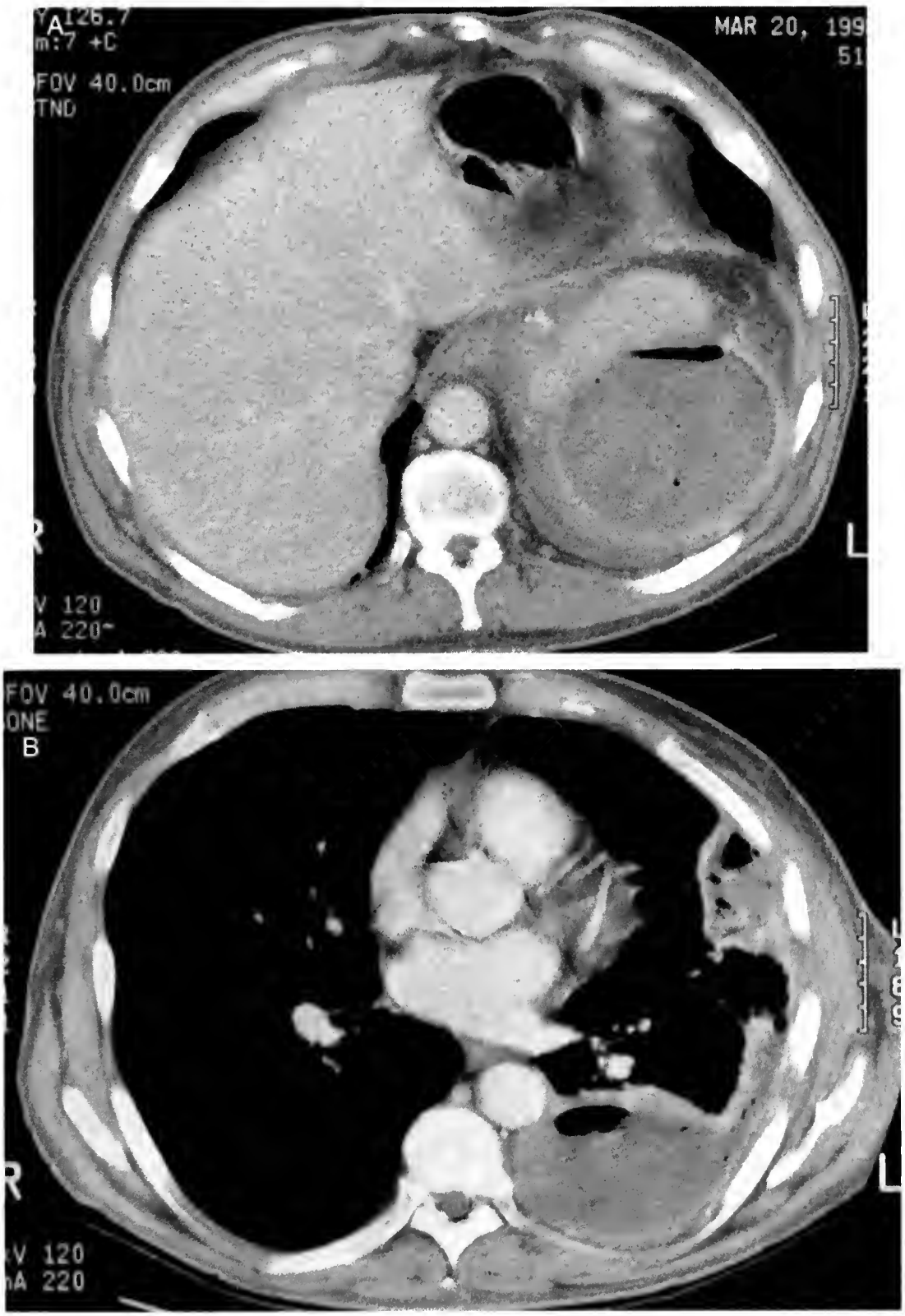


Fig. 6. Computed tomography (CT) detection and drainage of empyema and subphrenic abscess. A: Enhanced CT scan through the upper abdomen after gastric bypass surgery shows a left subphrenic abscess. B: CT through the lower thorax shows a multiloculated left pleural effusion, with air/fluid levels and enhancing margins representing empyema. C: Scan following placement of a subphrenic sump drain shows the catheter within the collection. D and E: Scans obtained with the patient in the right lateral decubitus position following placement of catheters into the posterior (D) and lateral (E) locules. The collections were all successfully drained. (*Figure continues on next page.*)

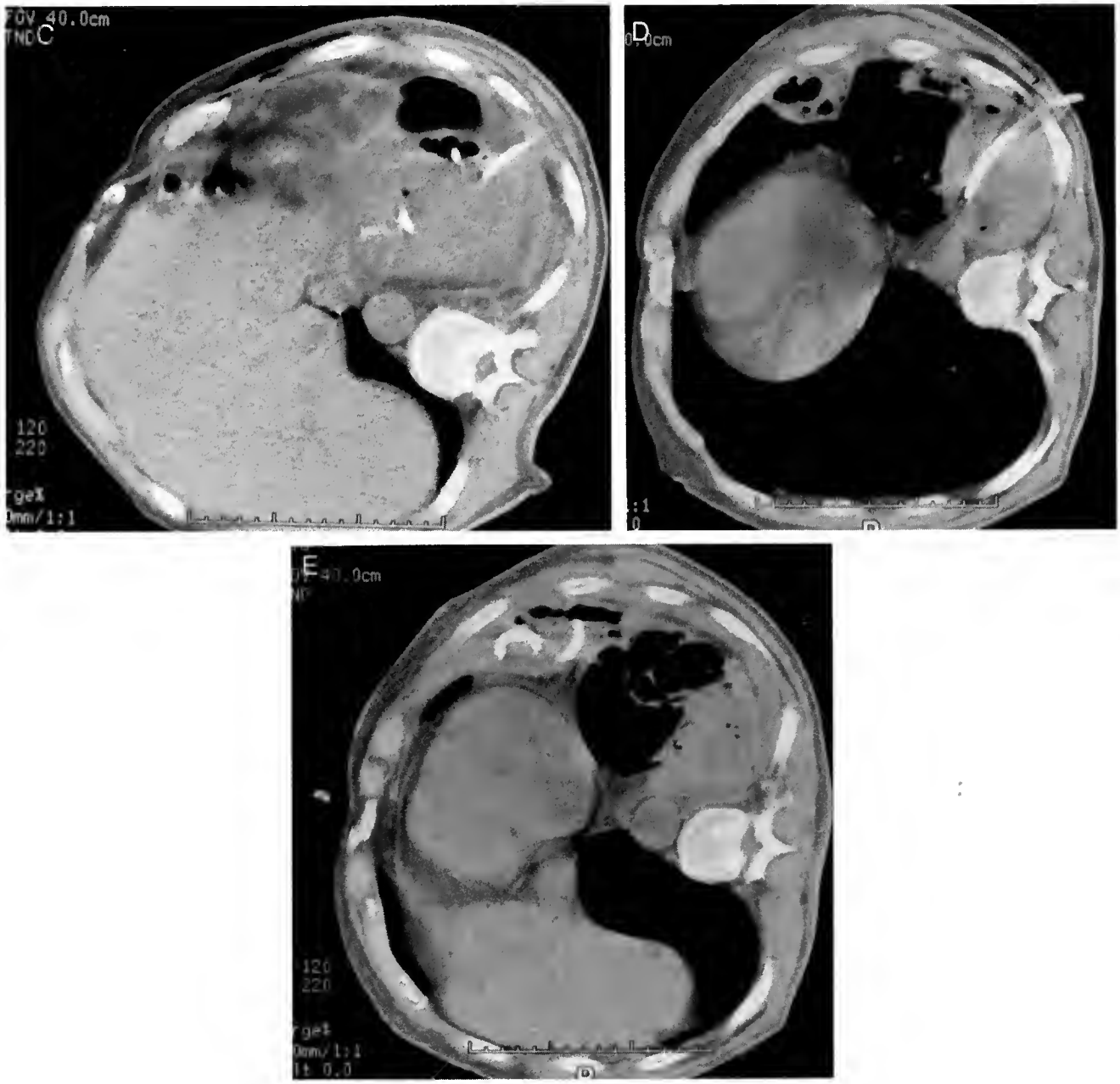


Fig. 6. *Continued*

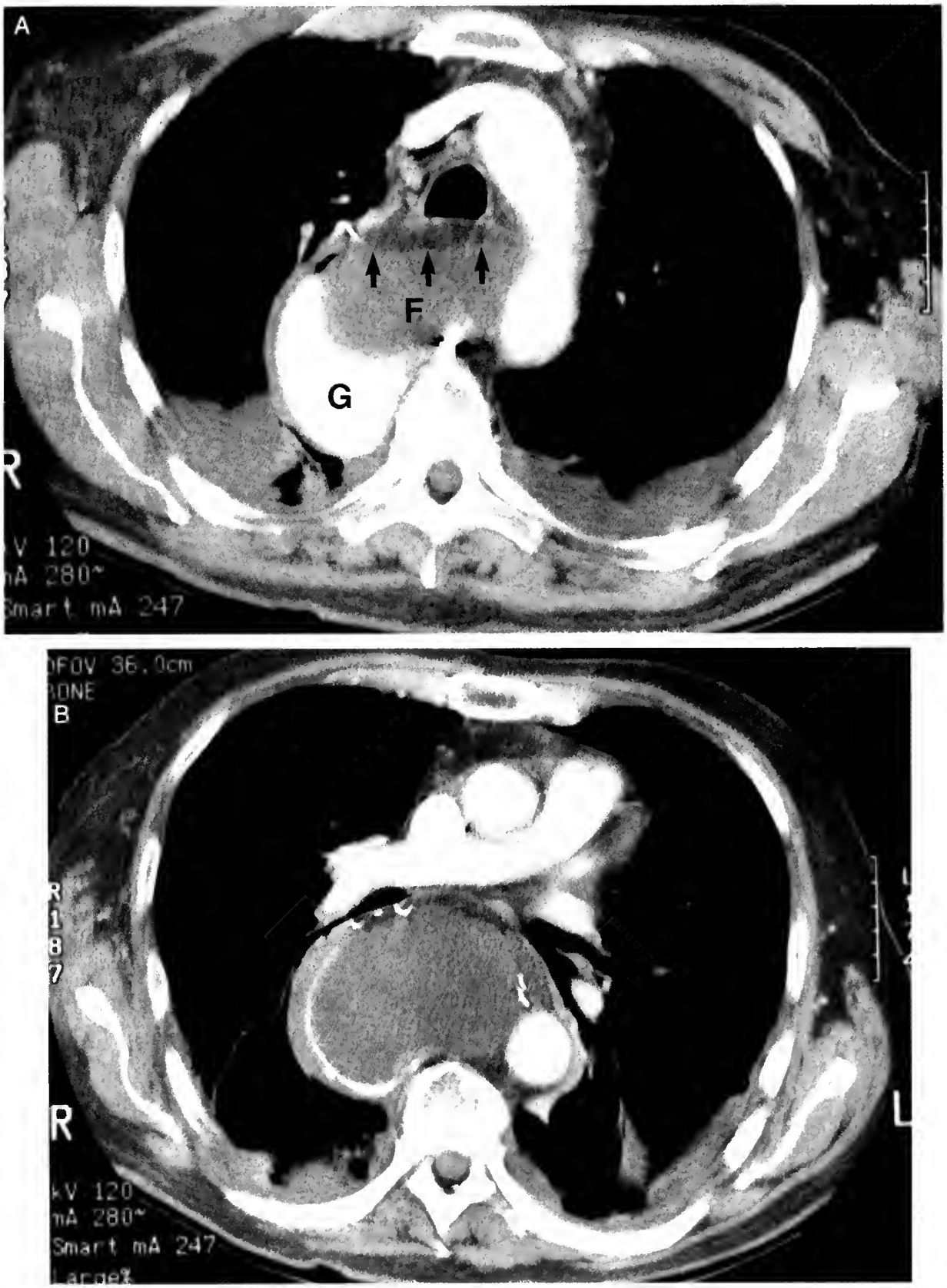


Fig. 7. (*Figure continues on next page; see next page for legend.*)

Fig. 7. Percutaneous drainage of post-esophagectomy mediastinal lymphocele. A and B: Computed tomography (CT) scans following oral and intravenous contrast administration in a 72-year-old man following esophagogastrectomy and gastric pull-through shows a postoperative mediastinal collection (F) containing a fluid/fluid level (small arrows). Note marked compression of the stomach in B. G = gastric pull-through. C: Scan following CT-guided catheter placement into mediastinal collection shows catheter in collection with the introduction of air (curved arrow). The high-attenuation material seen in the prespinal space is Ethiodol that has extravasated from the thoracic duct following pedal lymphangiography. The catheter retrieved 400 mL of chylous fluid.

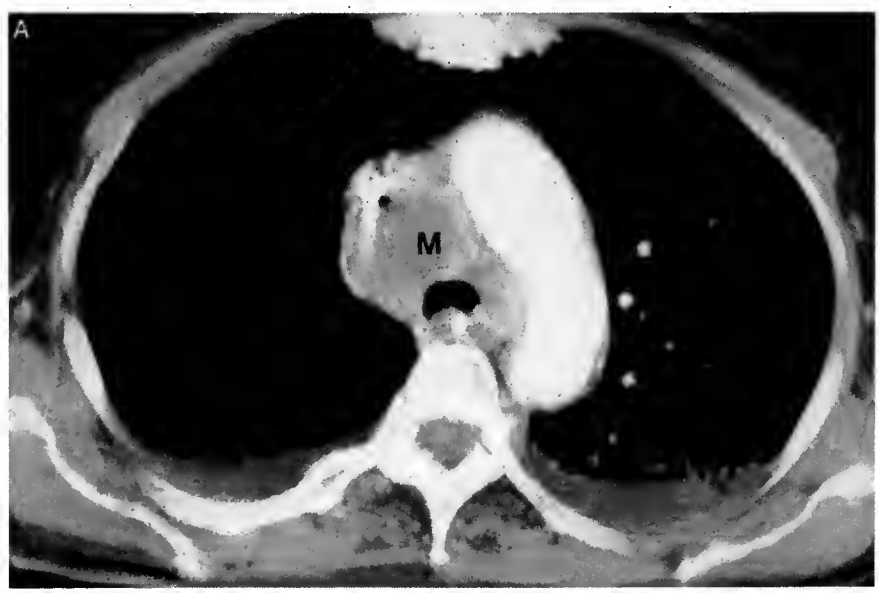
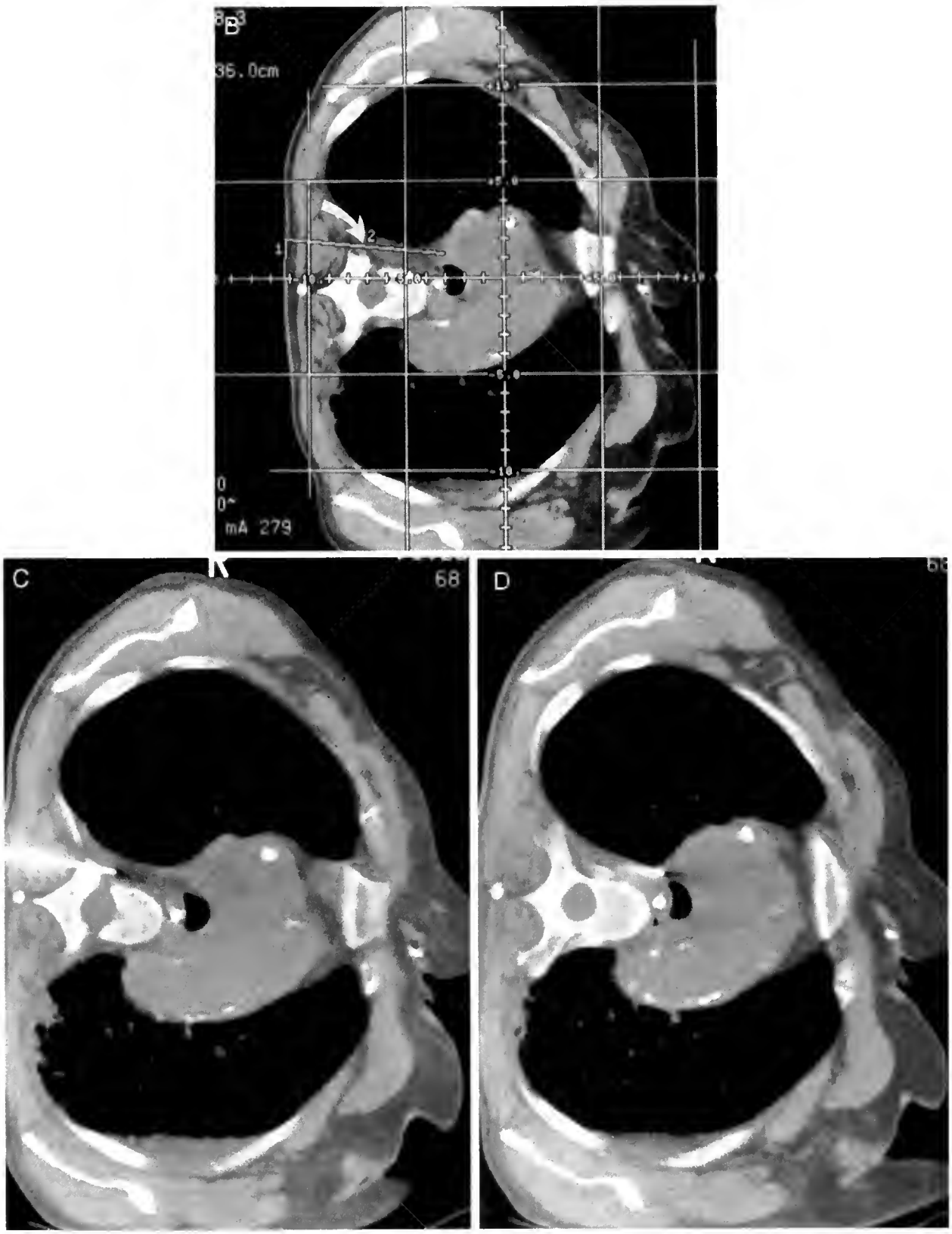


Fig. 8. Computed-tomography (CT)-guided mediastinal biopsy in postoperative patient. A: Enhanced CT scan at the level of the aortic arch after repair of an abdominal aortic aneurysm and an inability to wean from the ventilator shows a middle mediastinal mass (M). Note small bilateral pleural effusions. B: Scan with patient in left decubitus position shows anticipated biopsy route (curved arrow) via small right pleural effusion now repositioned to the paraspinal space. C and D: Scans obtained following needle placement show biopsy needle coursing through right pleural effusion to the edge of the mass. Fine-needle aspiration biopsy revealed small cell carcinoma that was unsuspected preoperatively. (*Figure continues on next page.*)



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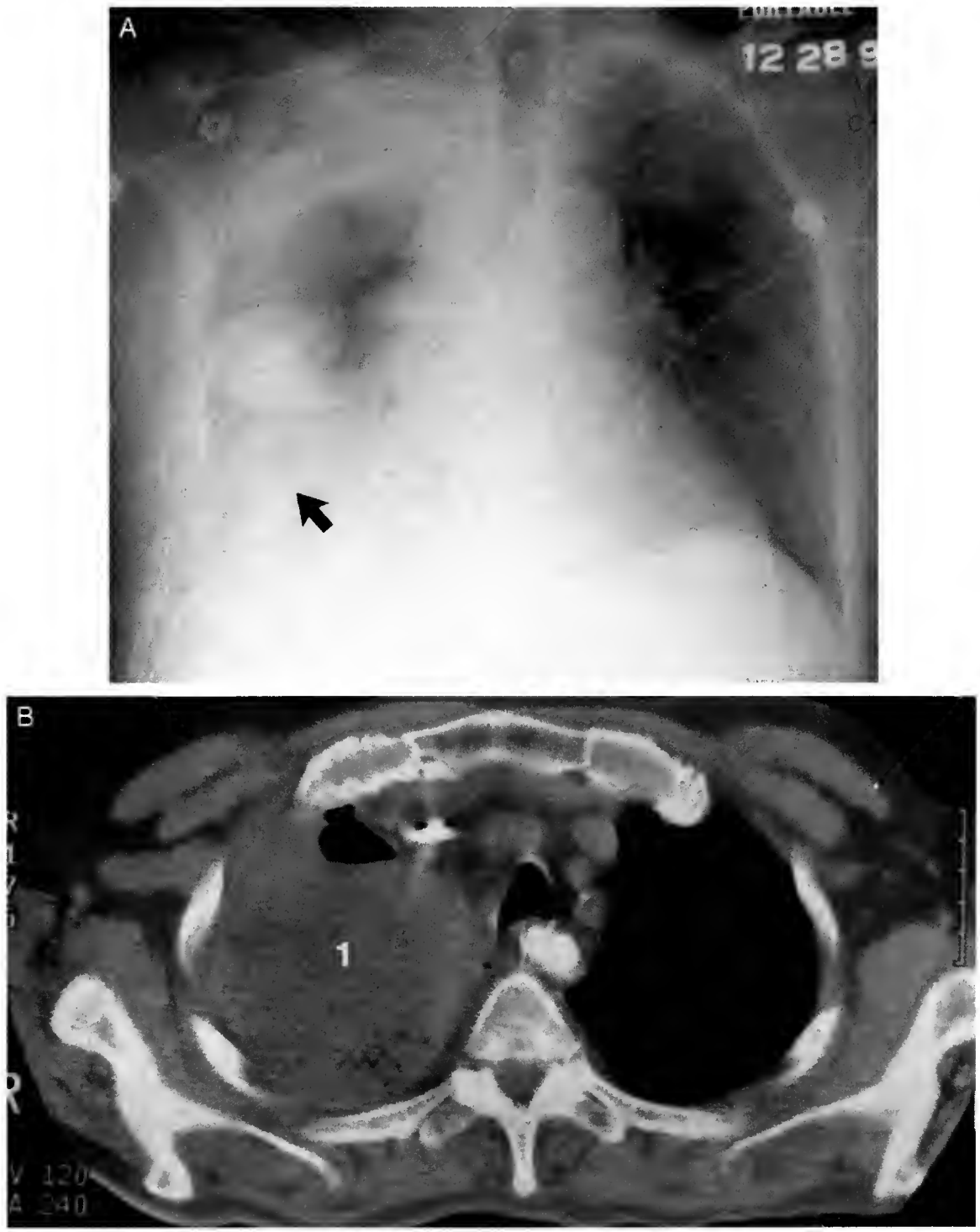
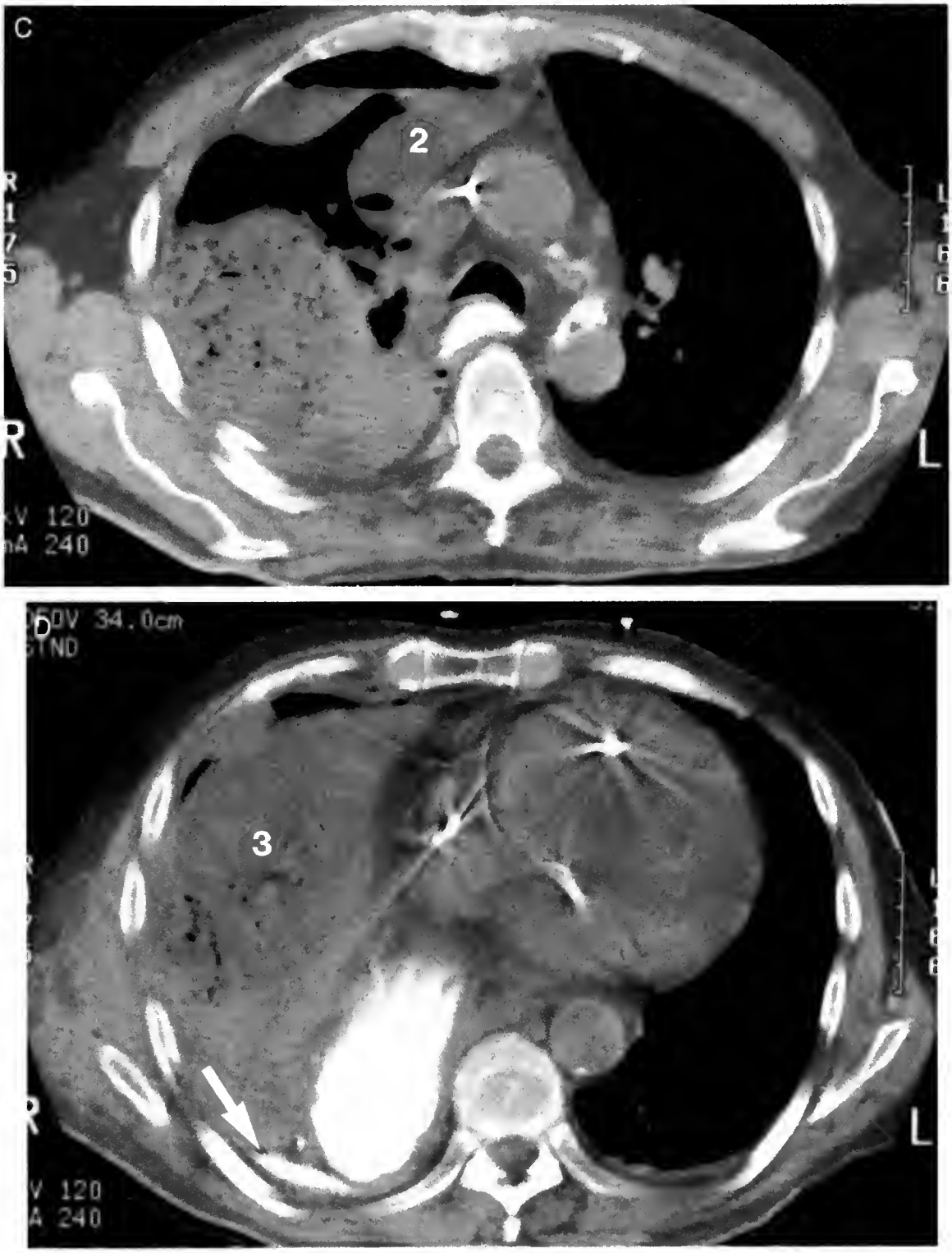


Fig. 9. Post-esophagectomy empyema drainage using intrapleural urokinase. A: Portable chest radiograph of a 58-year-old man 10 days after esophagectomy for esophageal carcinoma shows multiple large pleural collections despite the presence of a right chest tube (arrow). B through D: Scans obtained after administration of oral contrast show 3 collections: at the right apex (1 in 9B), within the anterior and medial right pleural space (2 in 9C), and subpulmonic (3 in 9D). Note the extravasated contrast (arrow in D) confirming a postoperative gastric leak. E through G: Scans obtained after placement of 3 drainage catheters show the catheters within the 3 collections. H: Portable chest radiograph after 5 days of urokinase administration shows marked resolution of the pleural collections. (*Figure continues on next page.*)



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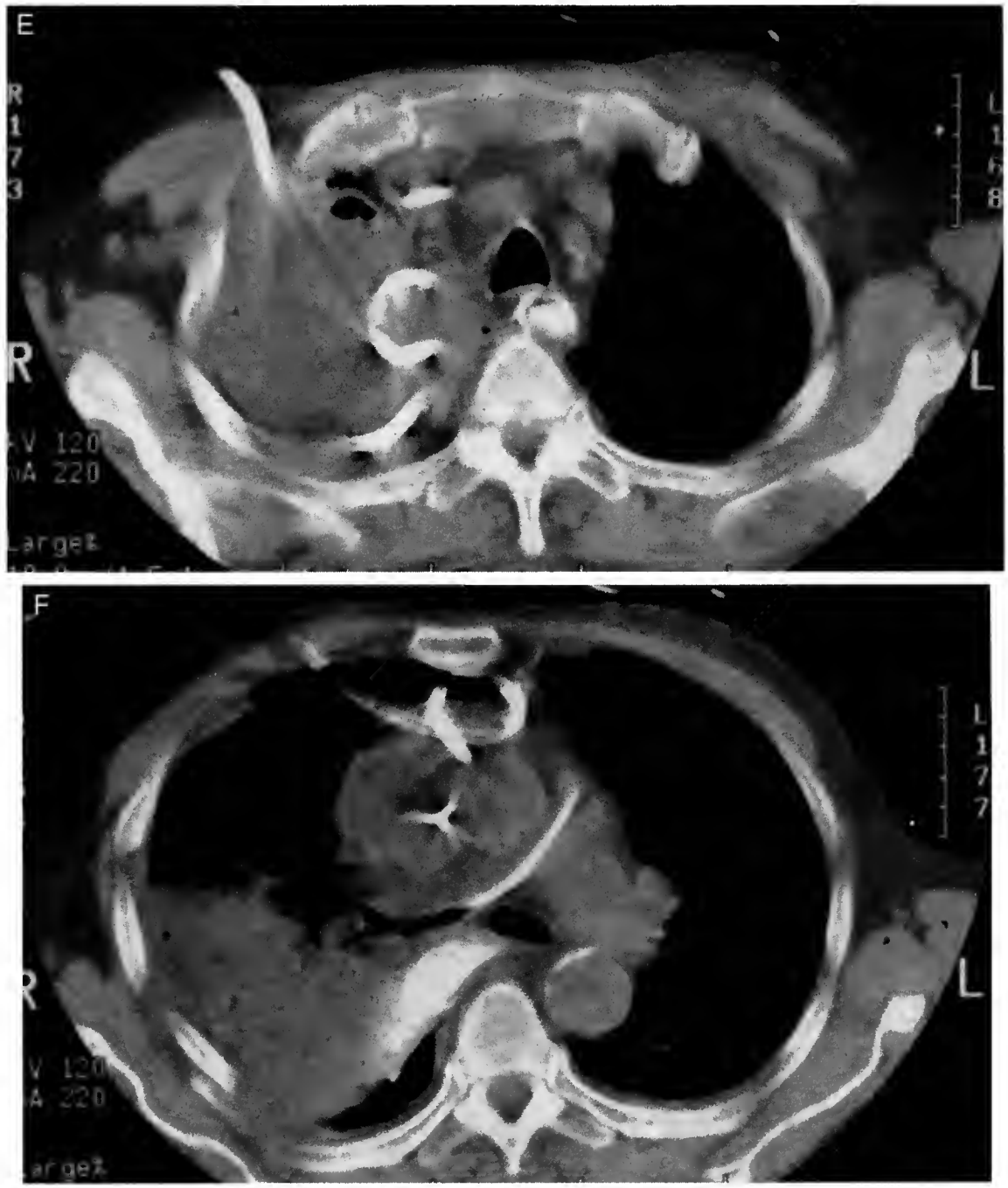

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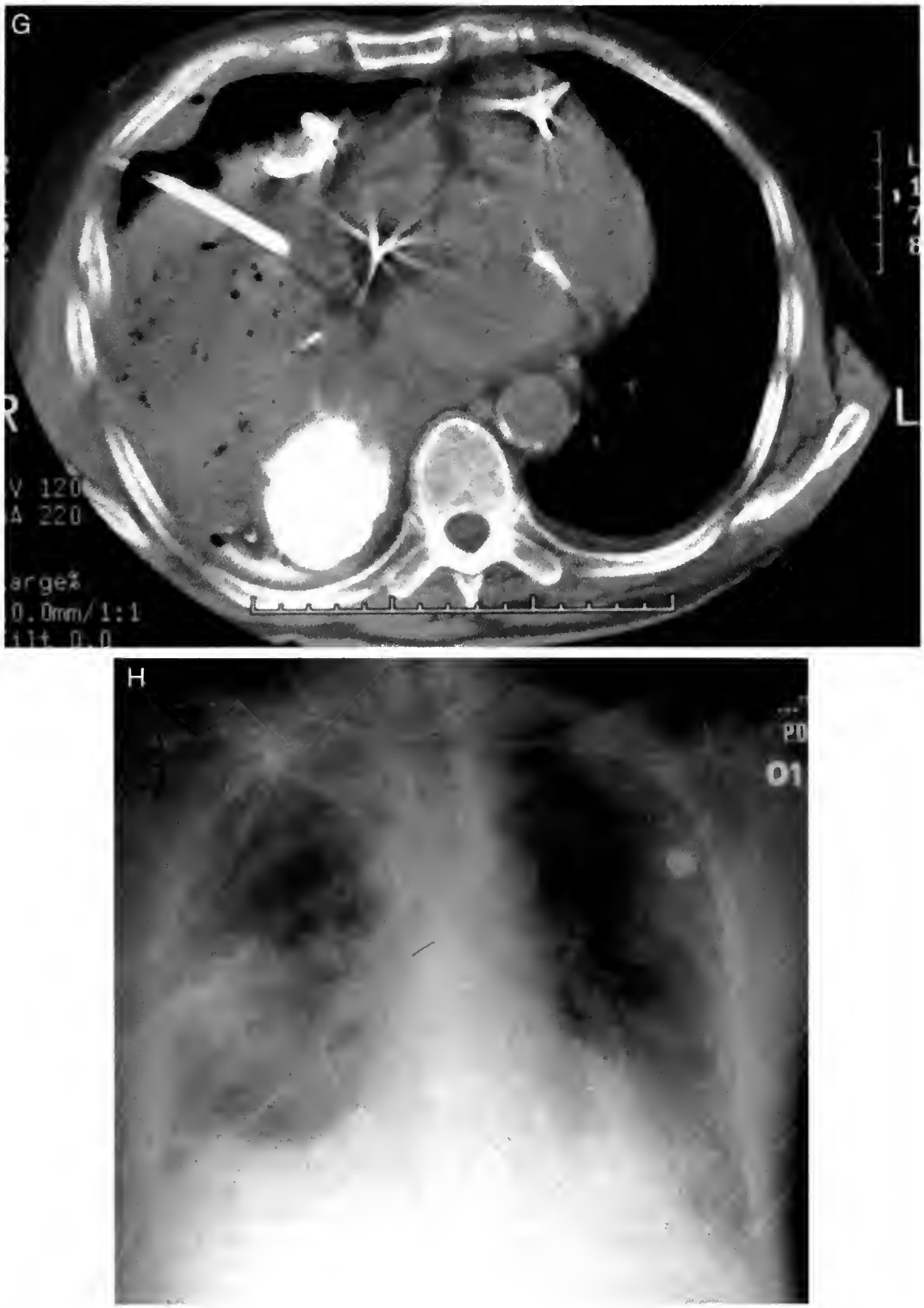

Fig. 9. *Continued*

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Chest Imaging in the Neonatal and Pediatric Intensive Care Units

George W Gross MD

Introduction
Technical Factors
Imaging Abnormalities in the Neonatal Intensive Care Unit
- **Endotracheal Tubes**
- **Enteric Catheters**
- **Umbilical Catheters**
- **Chest Tubes and Catheters**
- **Cannulae for Extracorporeal Membrane Oxygenation**

Medical Disorders
- **Respiratory Distress Syndrome**
- **Complications of Ventilatory Assistance for Respiratory Distress Syndrome**
- **Immature Lung Syndrome**
- **Patent Ductus Arteriosus**
- **Transient Respiratory Distress of the Newborn**
- **Meconium Aspiration Syndrome**
- **Amniotic Fluid Aspiration**
- **Neonatal Pneumonia and Sepsis**
- **Pulmonary Hemorrhage**
- **Pleural Effusion**
- **Chylothorax**
- **Atelectasis**

Surgical Disorders
- **Pneumothorax with Tension**
- **Congenital Diaphragmatic Hernia**
- **Congenital Cystic Adenomatoid Malformation**
- **Congenital Lobar Emphysema**

Imaging Abnormalities in the Pediatric Intensive Care Unit
- **Tubes, Lines, and Catheters**
- **Atelectasis**
- **Pneumonia and Complications**
- **Pulmonary Infection in the Immunocompromised Child**
- **Aspiration Pneumonitis**
- **Acute Smoke Inhalation**
- **Near-Drowning**
- **Hydrocarbon Aspiration and Pneumonitis**
- **Cardiac Failure and Pulmonary Edema**
- **Acute Chest Syndrome in Sickle Cell Disease**
- **Acute Respiratory Distress Syndrome**
- **Abnormal Pulmonary and Pleural Air and Fluid Collections**
- **Acute Chest Trauma**
- **Pulmonary Embolism**

Summary

[Respir Care 1999;44(9):1095–1126] *Key words: chest imaging, radiography, computed tomography, neonatology, pediatrics, intensive care unit respiratory failure, cardiac failure.*

Introduction

In both the neonatal intensive care unit (NICU) and the pediatric intensive care unit (PICU), thoracic imaging consists primarily of portable chest radiography. The typical exam consists of an anteroposterior (AP) projection only, usually because of the clinical acuity of patient illness or the lack of information likely to be provided by a lateral or other projection. However, additional projections occasionally provide satisfactory diagnostic evaluation; the lateral view is the most frequently added projection.

Because of its portability, ultrasonography of the thorax can be of value in collecting certain information or evaluating for certain problems, namely: the presence of loculations within and other characteristics of a pleural fluid collection; additional information regarding the thymus and other mediastinal structures; and, occasionally, the further characterization of chest wall abnormalities such as areas of swelling or potential masses. The field of echocardiography is beyond the scope of this article.

Although they provide superior diagnostic information in many disorders of the cardiovascular structures, radionuclide (nuclear medicine), magnetic resonance imaging (MRI), and computed tomography (CT) are not applicable to the child in either the NICU or PICU, because of the lack of portability of these imaging modalities.

Technical Factors

Portable chest radiography is typically performed using either a capacitor-discharge or battery-operated frequency-inverted mobile unit.[1] Proper radiograph exposure in the NICU typically involves settings in the 55–120 kilovolt range (usually around 60 kilovolts) and an exposure duration of 0.7–1.2 milliampere-seconds to avoid image degradation due to respiratory motion.[1] These exposure factors are progressively increased in the PICU, depending on patient size.[1]

The target (x-ray tube) to film distance is usually about 40 inches, shorter than the 72 inches typically used with conventional, nonportable radiography. This results in greater magnification effect, especially for structures farther from the radiographic film. For example, with the typical AP portable chest radiograph, a more anterior structure in the thorax, such as the heart, is magnified to a greater degree than a more posterior structure, resulting in apparent cardiomegaly, when heart size is actually normal.

For the portable chest radiograph obtained with a vertically-oriented x-ray beam (the most common orientation), personnel not shielded by lead aprons need be only about 12 inches from the primary x-ray beam in order to receive a radiation exposure at background level.[1] For angled or horizontally-aligned x-ray exposures, personnel should be to the side of or farther behind the patient in order to keep exposure to minimal, background levels.

Sonography of the thorax requires use of a coupling gel to provide direct contact of the sonographic transducer to the skin surface of the patient, in order to avoid any air gap that would terminate sound wave penetration and preclude obtaining meaningful imaging information. For similar reasons, portable sonography of the thorax is ineffective in penetrating bandages and wound sites with an associated air gap. Likewise, air-containing lungs cannot be evaluated with ultrasonography.

Imaging Abnormalities in the Neonatal Intensive Care Unit

Following live birth, the newborn rapidly exchanges intrapulmonary fluid for air, primarily by breathing. The initial 2–3 breaths result in nearly complete aeration of the lungs in most newborns.[2] In addition, some intrapulmonary fluid is cleared by lymphatic drainage or suctioning. The "cardiac" silhouette in the newborn actually consists of both thymus and heart, the former usually accounting for the preponderance of the cardiomediastinal margins. Radiographic signs of thymic tissue on the normal AP chest radiograph include "wavy" margins, the "notch sign," and the "sail sign." Because of thymic atrophy in many if not most ill neonates, the contribution of thymic tissue to the mediastinal configuration in these patients may be reduced, and even minimal.

The elevated in utero pulmonary vascular resistance progressively decreases within the first week of life. Persistence of this high pulmonary vascular resistance beyond the initial week may occur as an isolated phenomenon (termed "primary pulmonary hypertension") or in conjunction with a variety of cardiac or pulmonary disorders (termed "secondary pulmonary hypertension"). In the latter situation, the associated cardiac or pulmonary process determines the chest radiographic findings.

Virtually all NICU patients require life support that involves various catheters, tubes, and other devices. The component of these support items within or adjacent to the patient has characteristic radiographic appearances, preferred locations, and often orientations and configurations,

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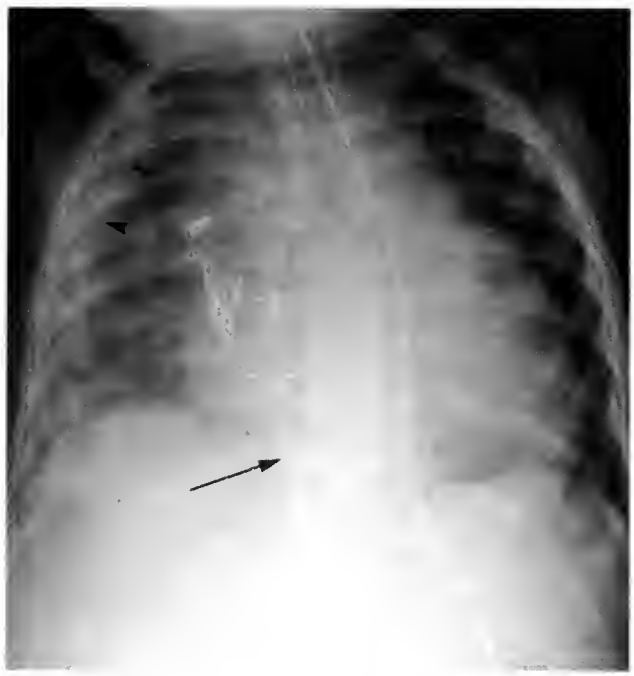

Fig. 1. Portable anteroposterior supine chest radiograph following cardiac surgery shows a mediastinal chest tube (arrow) entering from the sub-xiphoid region, a well-positioned right pleural chest tube, limited residual right pleural fluid (arrowheads), and well-positioned endotracheal and nasoenteric tubes. Also present are sternal wires, mediastinal surgical clips, and vascular coils for occlusion of collateral vascular shunt channels.

the evaluation of which is an important aspect of chest radiograph interpretation.

Endotracheal Tubes

All types of endotracheal tubes (ETTs) should be positioned so that the tip lies within the intrathoracic segment of the trachea and preferably at least 1 cm above the carina to allow for movement with change in patient respiration, head position, and other factors.[2–6] The tip of a well-positioned ETT in a newborn projects just below the medial clavicle ends on the AP chest radiograph.[2,3,6] Figures 1–4 illustrate satisfactory and unsatisfactory ETT positions in neonates.

Enteric Catheters

If used for decompression purposes, the polyethylene enteric catheter tip should be in the stomach; if for feeding purposes, in the third or fourth portion of the stomach, to minimize risk of perforation.[2] During tube passage, there is a risk of pharyngeal or esophageal perforation.[7] Figures

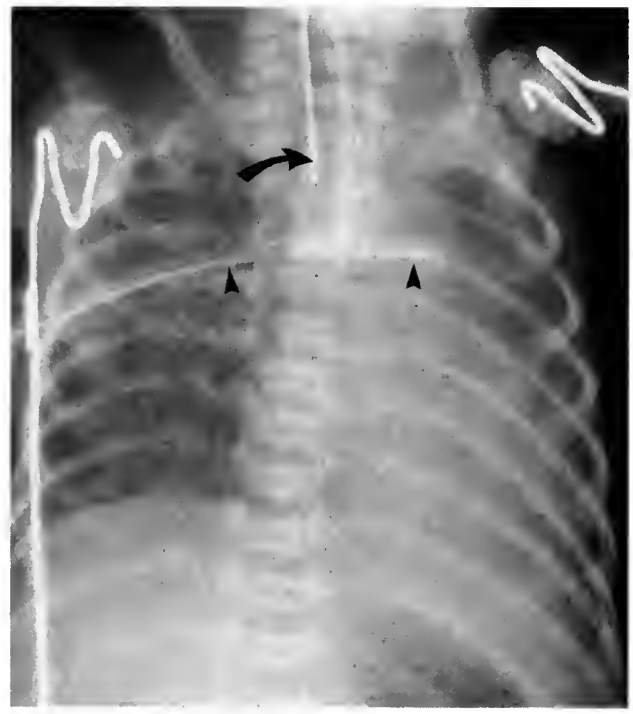

Fig. 2. Premature infant with respiratory distress syndrome on mechanical ventilatory assistance. The tip of the endotracheal tube is positioned too low, at the carina. The end of the nasoenteric tube (arrow) is in the upper thoracic esophagus, much too high. The single right pleural chest tube (arrowheads) placed for control of a prior pneumothorax extends well across the midline and may have penetrated into the left lung. There is relative increase in density in the left lower lung zone, which suggests partial left lower lobe atelectasis. (From Reference 3, with permission.)

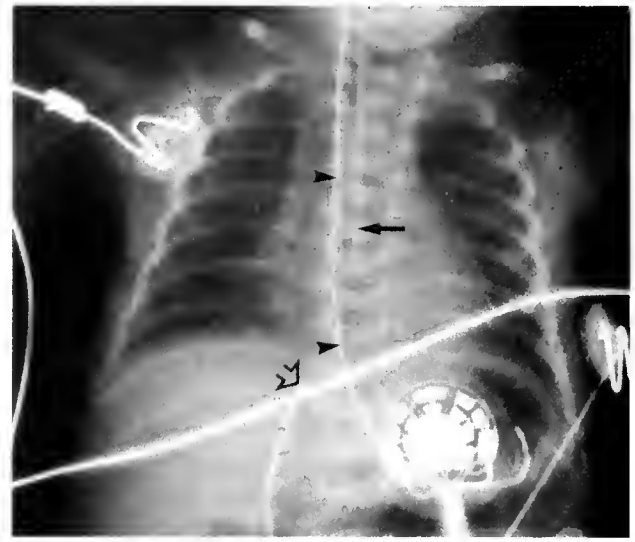

Fig. 3. Malpositioned endotracheal tube (ETT) in the esophagus. The precise location of the ETT (arrow) is unclear, but the fact that it extends well below the carina indicates that it cannot be situated in the trachea. Because it precisely parallels the well-positioned nasogastric catheter (arrowheads), the ETT was believed to be in the esophagus. The umbilical venous catheter tip (open arrow) is at the inferior vena cava-right atrial junction, in good position. (From Reference 3, with permission.)

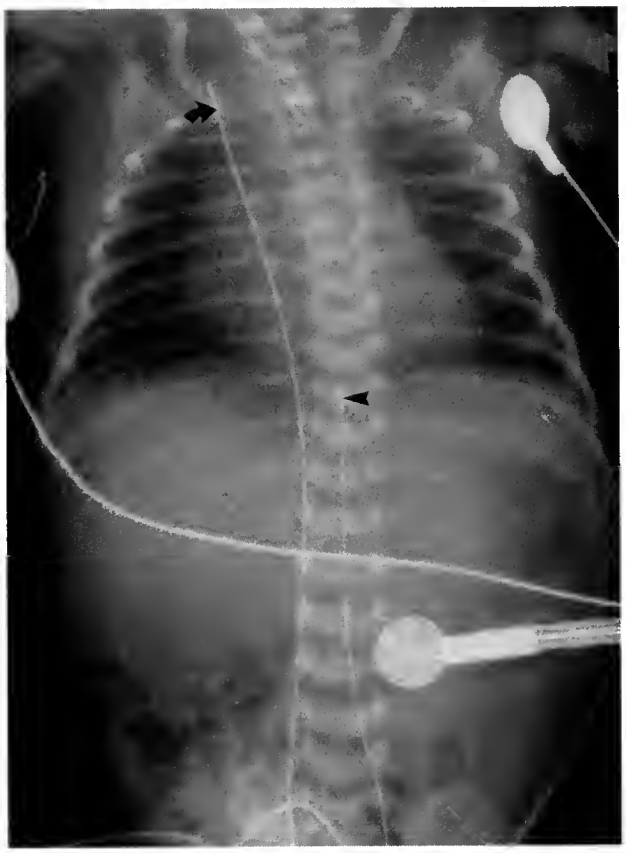

Fig. 4. Malpositioned umbilical catheters. The tip of the umbilical venous catheter (UVC) (arrow) extends up the superior vena cava, ending approximately in the internal jugular vein, much too high in position. The tip of the umbilical arterial catheter (UAC) (arrowhead) overlies the ninth thoracic vertebral body. This location would be satisfactory for a "high" UAC, but most neonatologists prefer the UAC to end in the region of L-3 to L-5. (From Reference 3, with permission.)

1–3 illustrate both satisfactory and unsatisfactory enteric catheter positions.

Umbilical Catheters

Passing through the umbilicus, umbilical artery, internal iliac artery, and common iliac artery prior to entering the aorta, the umbilical arterial catheter dips down into the pelvis on the AP and lateral abdominal radiographs before passing superiorly and posteriorly to reach the aorta.[7–11] Figures 4 and 5 illustrate satisfactory and unsatisfactory umbilical arterial catheter positions. Passing from the umbilicus to the heart, the umbilical venous catheter (UVC) traverses the umbilical vein, medial portion of the left portal vein, ductus venosus, left hepatic vein, and inferior vena cava.[11] On the radiograph the UVC passes directly superiorly and posteriorly to reach the heart.[8,11] Examples of normal and abnormal UVC tip position are shown in Figures 3–7.

Chest Tubes and Catheters

Most pleural chest tubes for drainage of pneumothoraces or pleural fluid collections have both an end hole and at least one side hole, all of which should be positioned within the pleural space (see Figs. 1, 2, and 6).[12,13] Perforation of the lung by a pleural catheter may occur in up to 25% of cases, and may be suggested on the chest radiograph by an increased density, representing hemorrhage or atelectasis around the tip of the tube.[7,13] Mediastinal chest tubes are usually employed following surgery for congenital cardiac disease or removal of mediastinal masses (see Fig. 1).

Cannulae for Extracorporeal Membrane Oxygenation

Extracorporeal membrane oxygenation (ECMO), a form of temporary life support utilized in children of various ages, is most commonly applied to neonates with severe and predictably fatal pulmonary diseases, usually with associated pulmonary hypertension. Dual (arterial and venous) cannulae are most frequently utilized. A single can-

Fig. 5. Malpositioned umbilical catheters. The umbilical venous catheter (arrow) extends to the liver, probably within the right portal vein. The umbilical arterial catheter (arrowhead) extends only to the midpelvis region and is probably near the junction of the umbilical and hypogastric arteries. (From Reference 3, with permission.)

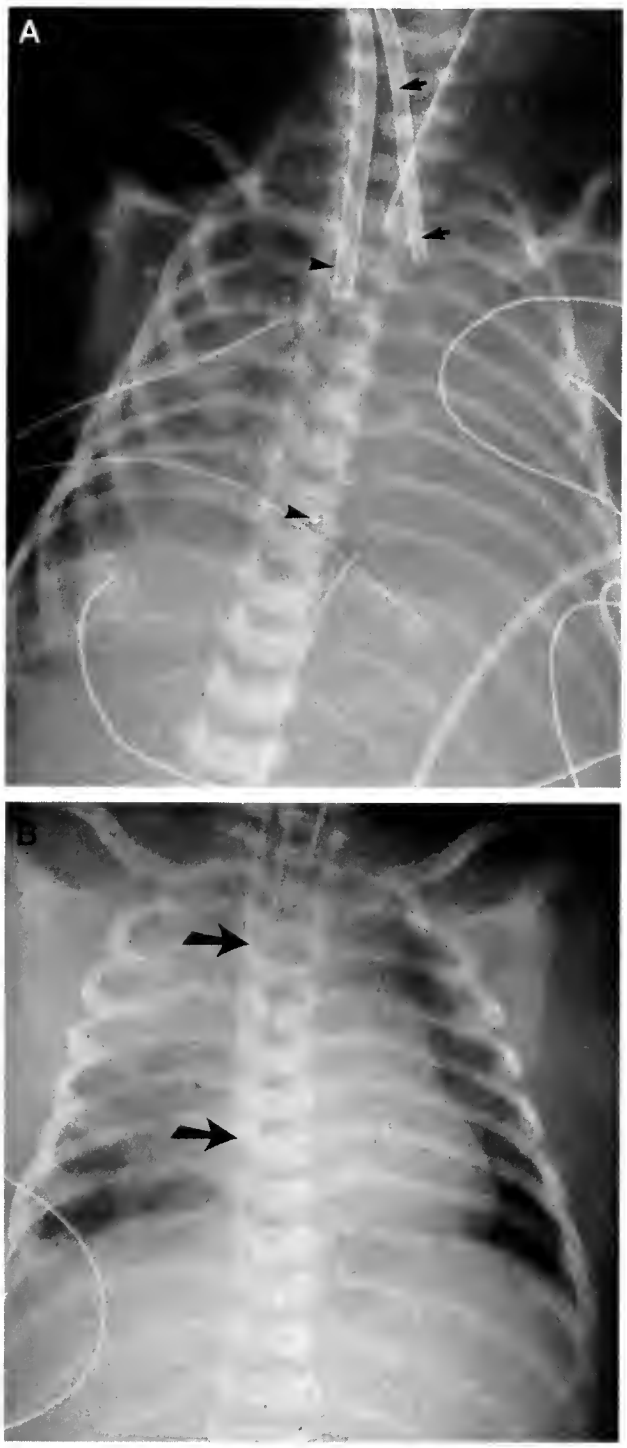

Fig. 6. Neonates with high predicted mortality and failing maximal medical and ventilatory support are potential candidates for extracorporeal membrane oxygenation (ECMO), a form of external life support. A: Carotid arterial (arrows) and jugular venous (arrowheads) cannulae are placed via a right neck incision into the aortic arch and right ventricle, respectively. Desaturated blood is removed via the jugular venous cannula, oxygenated in an external membrane oxygenator, and returned via the carotid arterial cannula. The lower end of the jugular venous cannula is marked by the opaque dot (lower arrowhead). Complete opacification of the lungs typically occurs early in the course of ECMO bypass support, but progressively resolves with improving pulmonary function. B: A minority of ECMO patients with satisfactory cardiac function may be on dual-lumen, single cannula bypass (arrows), which avoids the need to surgically violate the carotid artery. Note the satisfactorily positioned endotracheal tube, umbilical venous catheter, and right chest tubes. (From Reference 3, with permission.)

nula with dual lumen is less commonly used.[14,15] Figure 6 shows examples of ECMO cannulae.

Of the vast number of potential disorders of the newborn chest, only the more prevalent will be discussed. Historically, these disorders have been divided into "medical" and "surgical" entities, based on the primary approach to their management.

Medical Disorders

Respiratory Distress Syndrome

A manifestation of pulmonary immaturity and surfactant deficiency, respiratory distress syndrome (RDS) occurs predominantly in premature newborns under 35 weeks gestational age and 2.5 kg birthweight.[2,3,7,16,17] The hallmark pathologic finding in RDS is acinar atelectasis, which manifests on the chest radiograph as hypoventilated lungs with a diffuse granular or reticulogranular lung opacification with air bronchograms (see Fig. 7).[2,3,7,16,18–20] Patients with RDS are usually symptomatic shortly after birth, and the chest radiograph is abnormal within 12–24 hours.[2,3,16] If they are placed on mechanical ventilatory assistance, and depending on ventilatory pressure settings, the lungs may become more lucent and normally expanded, or even hyperinflated.[2,3,7]

Complications of Ventilatory Assistance for Respiratory Distress Syndrome

The neonate with more severe RDS who requires mechanical ventilatory assistance is at risk for a variety of "air block" complications related to the underlying pulmonary disease process and the associated increased airway pressure of mechanical ventilation.[2,3,7,16,21,22] When alveolar rupture occurs, air can accumulate in the peribronchial and perivascular spaces to produce pulmonary interstitial emphysema (PIE), which can appear on the chest radiograph as small round, linear, or branching lucencies that may extend to the periphery of the lungs[2,3,7,16,21–26] (Figs. 7–9). Extension of PIE to the lung periphery can produce subpleural blebs and rupture into the pleural space, resulting in pneumothorax (Figs. 8–10).[2,3,7,16,27–33] Progressive accumulation of air in the pleural space may result in pleural space expansion (tension pneumothorax), in turn producing contralateral me-

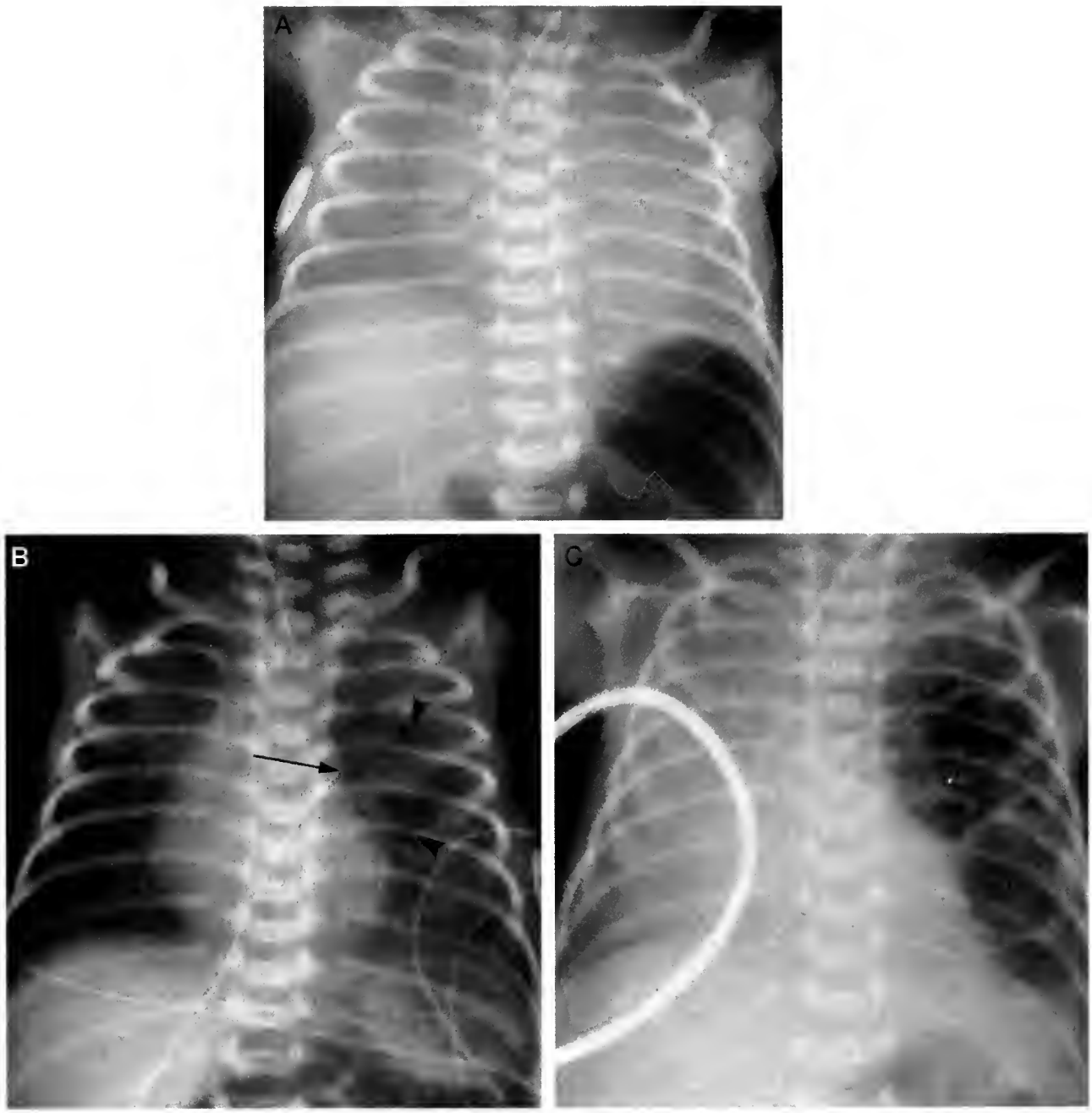

Fig. 7. A: Anteroposterior portable chest radiograph of a premature newborn with severe respiratory distress syndrome (RDS) and on mechanical ventilation shows diffuse pulmonary granularity consistent with the clinical diagnosis of RDS. No complication of ventilatory assistance is present. B: Two days later, a central lucency (arrowheads) suggests interstitial pulmonary air in the left lung. Note the tip of the umbilical venous catheter in a left upper lobe pulmonary vein (arrow). Endotracheal tube position is satisfactory. C: By 13 days of age, overexpansion of the left lung, with coarse cystic change, suggests bronchopulmonary dysplasia. A relative increase in density of the right lung may partly reflect compressive atelectasis. No pneumothorax occurred in this neonate. (From Reference 3, with permission.)

diastinal shift, depression of the ipsilateral diaphragm, and collapse of the adjacent lung (Figs. 8 and 11).[3,7,16,21] Extension of PIE centrally can result in pneumomediastinum (Figs. 9 and 12), pneumopericardium (Fig. 13), or, with caudal dissection, either retroperitoneal air (see Fig. 9) or a pneumoperitoneum.[2,3,7,34–38] Alternatively, air entering the lung interstitium via alveolar rupture may coalesce into a pseudocyst (Figs. 8 and 14).[2,39,40]

Bronchopulmonary dysplasia (BPD), or chronic lung disease of prematurity, is a common complication in newborns requiring ventilatory assistance.[2,41–45] Thought to reflect a combination of oxygen toxicity and barotrauma,

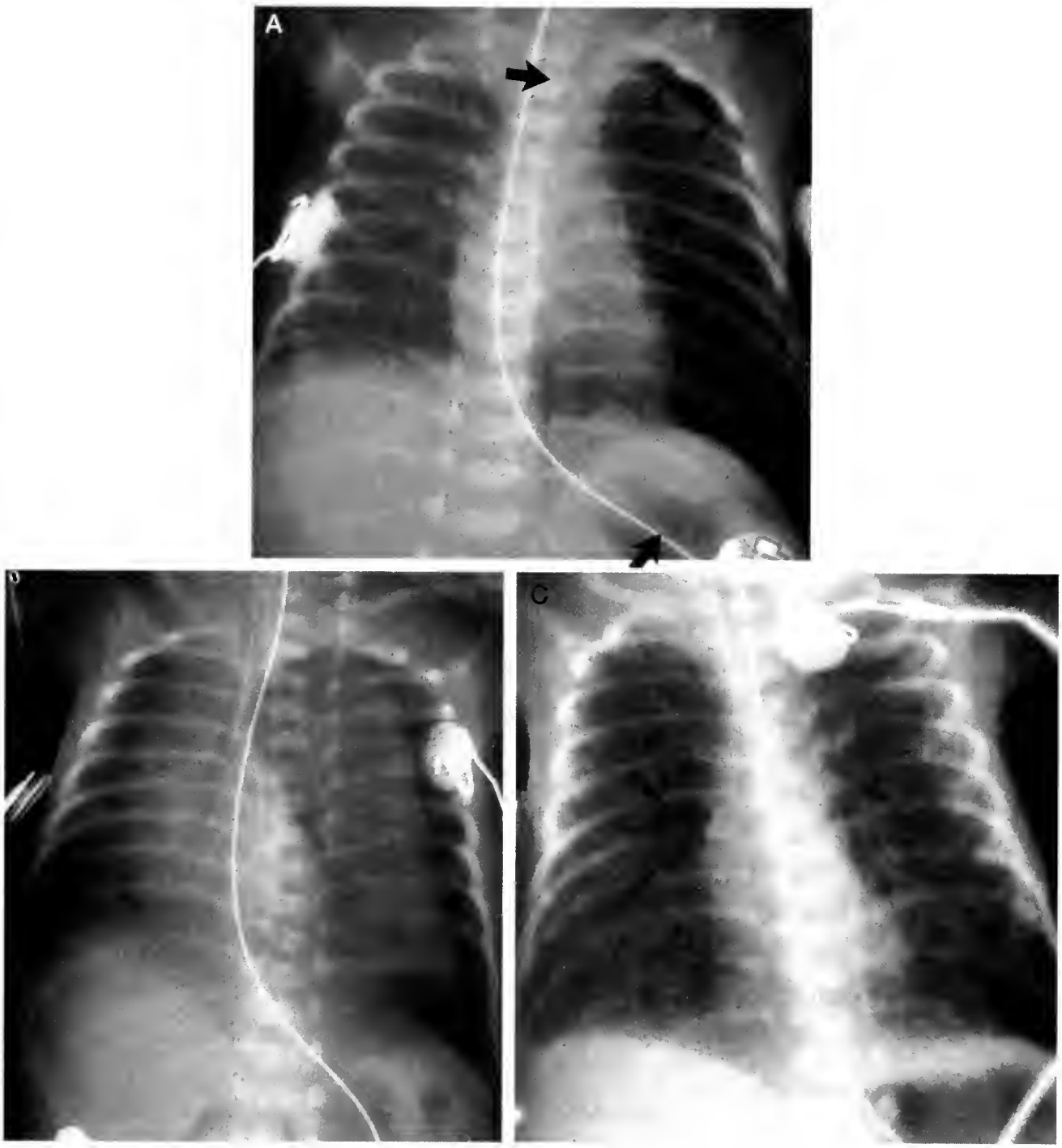

Fig. 8. A: Anteroposterior (AP) portable chest radiograph in a 2-day-old premature infant with respiratory distress syndrome (RDS) shows diffuse increase in pulmonary density, compatible with RDS, and accompanied by diffuse pulmonary interstitial emphysema (PIE). In addition, a left pneumothorax (arrowheads) is present. The pneumothorax tends to collect anterior to the lung in these supine neonates, and is usually larger than suggested by the typical AP supine chest radiograph. Both the endotracheal and nasogastric tubes (arrows) are in good position. B: Repeat chest radiograph on same day as Fig. 8A, and following sudden clinical deterioration, shows enlarged left pneumothorax, now with a tension component. Note the fine cystic changes within the lungs, best seen on the left, reflecting PIE. C: Four days later there is marked hyperinflation of both lungs, with a coarse appearance to the lung parenchyma, reflecting worsening PIE. Overlying the right hilum is a larger, focal pulmonary cyst (arrows). The left pneumothorax had been aspirated and did not recur. (From Reference 3, with permission.)

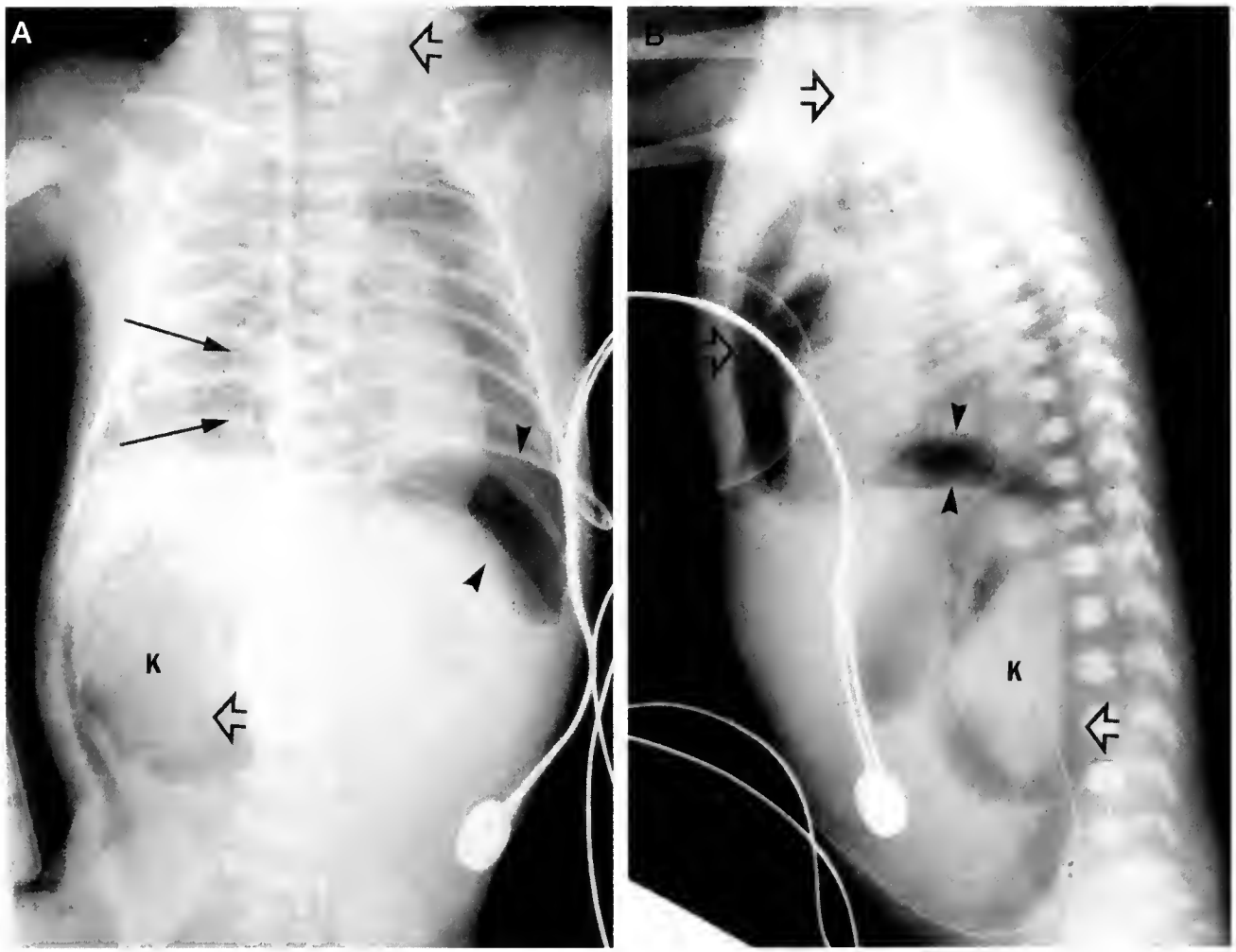

Fig. 9. Premature newborn with severe respiratory distress syndrome and on assisted ventilation. A: Anteroposterior radiograph. B: Lateral radiograph. Both views show predominantly right-sided pulmonary interstitial emphysema (arrows), a left pneumothorax (arrowheads), and a pneumomediastinum with air tracking up into the neck and down into the retroperitoneum (open arrows), surrounding the right kidney (K). (From Reference 3, with permission.)

BPD was initially described as consisting of 4 stages.[41] Stage I appears identical to RDS, with bilateral fine granular density, air bronchograms, and hypoaerated lungs.[2,3,7,41–43,45–47] Stage II BPD, developing between 4 and 10 days of age, appears as a "white out" of the lungs, which may be largely a reflection of an associated left-to-right shunt via a patent ductus arteriosus (PDA).[2,6,41–43,46,47] In Stage III BPD, developing between 10 and 20 days of age, the lungs have a bubbly appearance (see Fig. 7).[2,3,7,41–43,46,47] Stage IV BPD, in neonates over one month of age, is a combination of bubbly cystic lung changes and hyperinflation (Fig. 15).[2,3,7,41–43,45–47]

Both the clinical and radiologic courses of BPD frequently differ from the original pattern described, with most patients currently not manifesting radiologic Stage II but instead developing a mixed pulmonary pattern of atelectasis alternating with areas of emphysema.[7]

Immature Lung Syndrome

Occurring in small premature neonates under 1,500 g, immature lung syndrome differs from RDS in that symptoms of respiratory distress are usually delayed for 4–7 days after birth, and overall survival is better than with RDS.[2] The chest radiographic findings differ from those of RDS in that the lungs are not under-expanded, the diffuse pulmonary granularity of immature lung syndrome has limited or no associated air bronchograms, and the onset of radiographic findings is usually delayed for several days.[2,48–50]

Patent Ductus Arteriosus

Failure of the ductus arteriosus to close in the days following birth, in conjunction with the typical decrease in

Fig. 10. Relative lucency of the medial half of the right lung field, compared with the lateral portion, is an abnormal appearance and suggests a pneumothorax with the pleural air localizing medially and anteriorly. Generalized pulmonary granularity with air bronchograms reflects respiratory distress syndrome. The pattern of density of the lungs on an anteroposterior supine chest radiograph should not show an increase in lucency progressing from the lateral lung margin to the mediastinum; if present, pneumothorax or pneumomediastinum must be considered. (From Reference 3, with permission.)

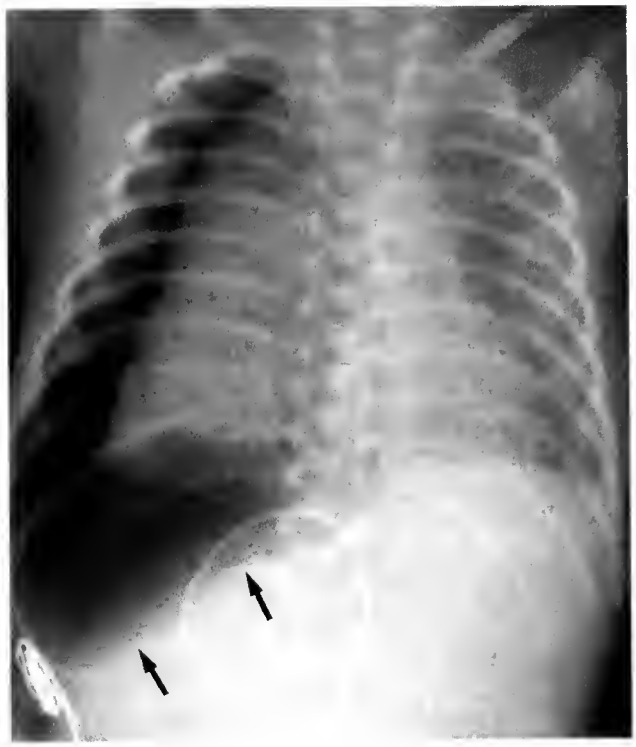

Fig. 11. Premature newborn with respiratory distress syndrome (RDS) and on mechanical ventilatory assistance. In addition to diffuse pulmonary granularity reflecting the atelectasis of RDS, there is a right tension pneumothorax, with depression of the right diaphragm (arrows), partial collapse of the right lung, and mild mediastinal shift to the left. (From Reference 3, with permission.)

pulmonary vascular resistance that normally occurs, can result in a left-to-right shunt via PDA.[2,3,7,51–53] With small PDAs, the chest radiograph will be normal or reflect an associated lung disease process, while moderate to large PDAs will show increased opacification of the lungs accompanied by increased heart size (Fig. 16).[2,3,7,51,53]

Transient Respiratory Distress of the Newborn

Transient respiratory distress of the newborn (TRDN), also known as wet lung disease, is a common cause of transient and usually relatively mild respiratory distress in the newborn, especially if born by cesarean section.[2,3,7,16,54–55] The most common radiographic findings are of accentuated interstitial lung markings, limited pleural fluid collections, and mild cardiomegaly (Fig. 17).[2,3,7,16,54–55] More severe cases can suggest pulmonary edema, with patchy, confluent areas of lung opacity. These patients seldom require intubation, and the symptoms and radiographic findings usually resolve within 24–48 hours.[2,3,7,16,54–55]

Meconium Aspiration Syndrome

Reflecting the intrauterine or intrapartum aspiration of meconium-stained amniotic fluid, meconium aspiration syndrome varies clinically and radiographically, depending on the severity of meconium aspiration.[2,3,7,16,56–59] The usual chest radiographic findings with meconium aspiration syndrome are generalized pulmonary hyperinflation (often severe) accompanied by bilateral, patchy, irregular infiltrates (Fig. 18).[2,3,7,16,56–59] Air-block complications (especially pneumothoraces) are seen in 25% of patients with meconium aspiration syndrome.[2,7,57] Pulmonary hypertension and right-to-left shunt across the patent foramen ovale and/or PDA is a common associated problem.[16,57,58]

Amniotic Fluid Aspiration

Aspiration of nonmeconium-stained amniotic fluid by the fetus prior to or during delivery can result in a type of chemical pneumonitis, with airway obstruction due to aspirated debris and accompanied by pulmonary hyperinflation.[2] The radiographic findings are generally similar to meconium aspiration syndrome, except for a tendency for more rapid clearing (Fig. 19).[16]

Neonatal Pneumonia and Sepsis

The chest radiographic findings in neonatal pneumonia are highly variable, generally nonspecific, and depend in

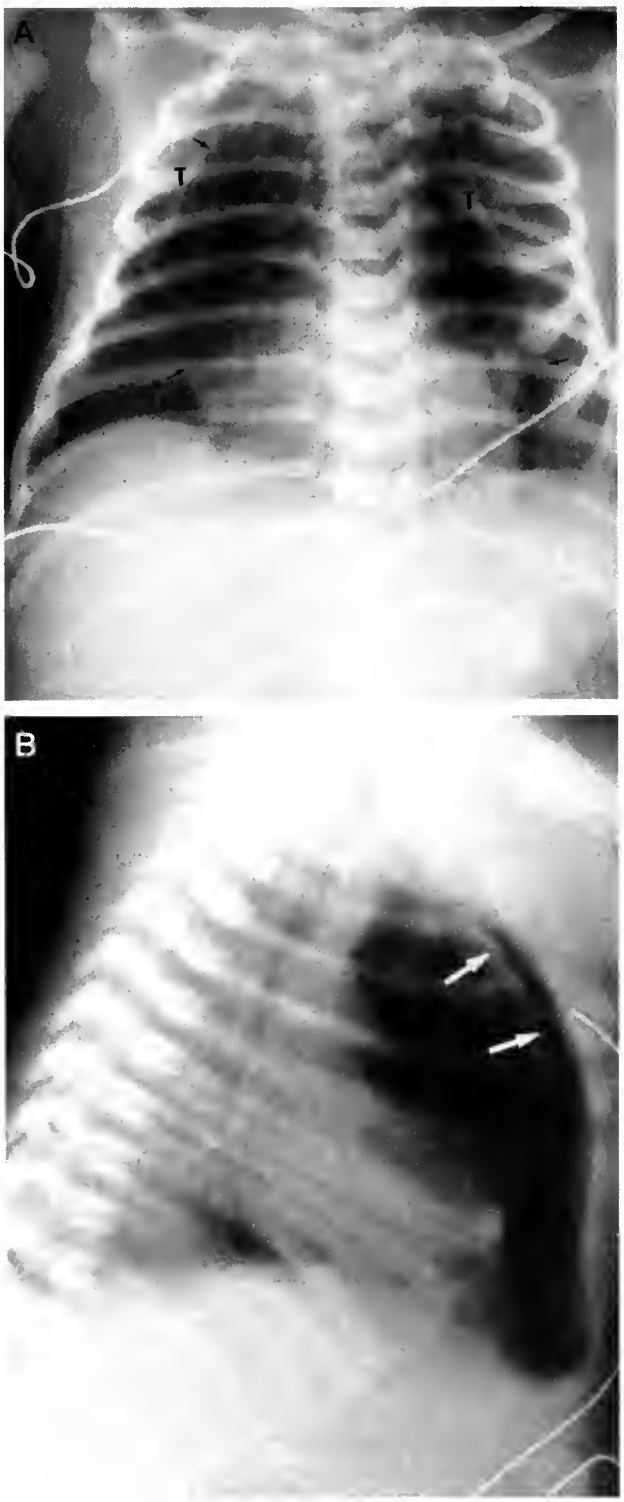

Fig. 12. A: A large pneumomediastinum (arrows) in a newborn with respiratory distress caused by meconium aspiration. The right and left lobes of the thymus (T) are silhouetted medially and laterally by air in the mediastinum and lungs, respectively. B: Lateral view shows mediastinal air anteriorly, with isolation of the thymus (arrows). Pneumomediastinum is usually not clinically important, and rarely requires intervention. (From Reference 3, with permission.)

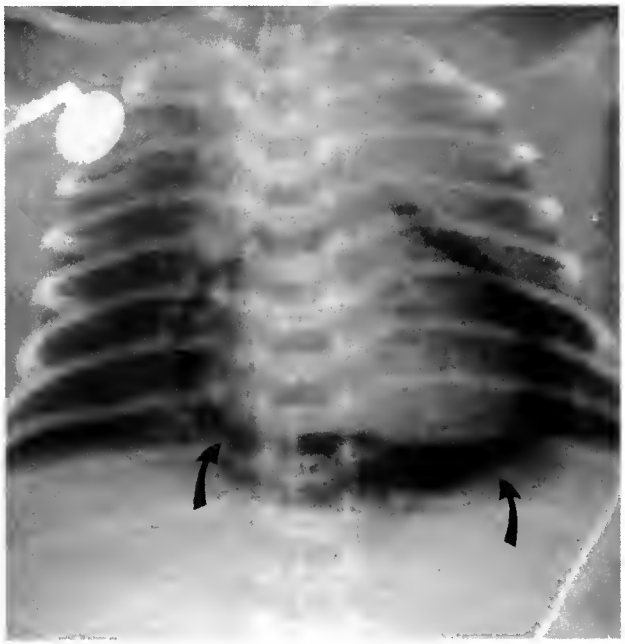

Fig. 13. Two-day-old infant with respiratory distress syndrome and on assisted ventilatory support. Anteroposterior chest radiograph after sudden clinical deterioration shows the heart to be silhouetted by a zone of lucency reflecting a pneumopericardium (arrows). Unlike pneumomediastinum, a pneumopericardium will have air extending around the heart, including the inferior margin. (From Reference 3, with permission.)

part on the etiology.[16,59–64] The most common chest radiographic appearance of neonatal pneumonia, from a variety of organisms, is one of patchy, asymmetric pulmonary opacities, accompanied by hyperinflation of the lungs (Fig. 20).[2,16] Clinical suspicion of pneumonia should increase if hyperinflation and patchy infiltrates are accompanied by pleural effusions.[2] Empyema associated with neonatal pneumonia would suggest either *Staphylococcus* or *Klebsiella* etiology.[2] The development of pneumatoceles with therapy increases the likelihood of staphylococcal pneumonia (Fig. 21).[2] Group B streptococcal pneumonia produces reticulogranular densities identical to those seen with RDS, but is much more likely to have associated pleural effusions and pulmonary hyperaeration, which are uncommon in uncomplicated RDS (Fig. 22).[2,3,7,16,65–67]

Pulmonary Hemorrhage

Pulmonary hemorrhage in the newborn is usually due to capillary damage resulting from severe hypoxia.[7] Although uncommon, acute pulmonary hemorrhage can result in rapid development of respiratory distress, accompanied by bilateral patchy infiltrates or complete pulmonary opacification on the chest radiograph, depending on the severity of the pulmonary hemorrhage (Fig. 23).[2,3,7,68] Blood or sero-

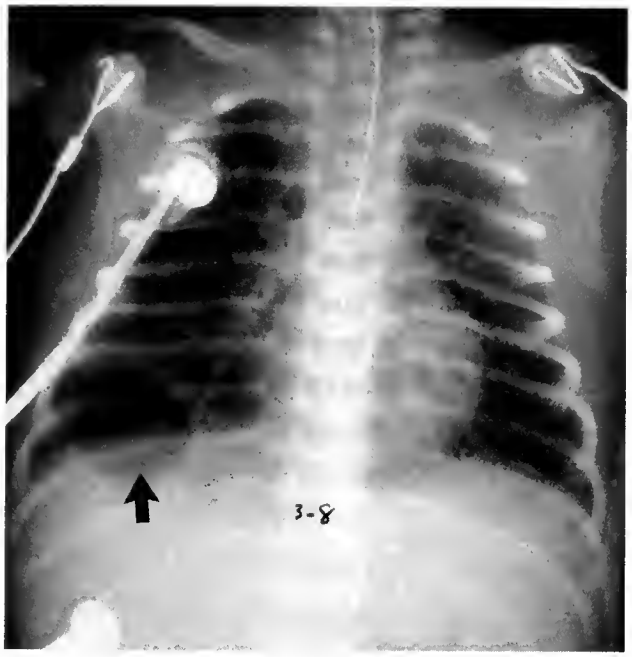

Fig. 14. Three-day-old infant with respiratory distress syndrome and on mechanical ventilation. In addition to generalized pulmonary interstitial emphysema bilaterally, a large parenchymal cyst (arrow) has developed at the right lung base. Spontaneous rupture of these cysts can result in the sudden development of a pneumothorax. (From Reference 3, with permission.)

sanguinous fluid emanating from the nose, mouth, or ETT is the usual clinical indication.[3,7,16]

Pleural Effusion

Congestive heart failure, erythroblastosis fetalis, idiopathic or iatrogenic hypervolemia, obstruction of pulmonary venous return, and transient tachypnea of the newborn are among the many potential causes of pleural effusion in the newborn.[2,3,16] Since the neonate in the NICU is typically maintained in the supine position, and pleural fluid tends to accumulate in the dependent portion of the chest, smaller amounts of pleural fluid may produce no radiographic change, or simply a diffuse increase in opacity of the involved hemithorax.[3] Large quantities of pleural fluid will separate the lucency of aerated lung from the lateral chest wall by a zone of homogeneous soft tissue density (see Fig. 1).[3] A decubitus AP chest radiograph (the side of the chest containing the pleural fluid being dependent) will better show the presence and extent of pleural fluid.[3]

Chylothorax

Chylothorax is the most common cause of a large pleural fluid collection (usually on the right side) in the newborn and most likely reflects rupture of the thoracic duct or transient central venous hypertension during delivery (Fig. 24).[2,16] Treatment can consist of conservative management, repeated thoracenteses or, if particularly severe, chest tube drainage.[2,16]

Atelectasis

Loss of lung volume, or atelectasis, is a common occurrence among neonates in the NICU and is usually due to airway obstruction from retained secretions, aspiration, or malposition of the ETT.[3] Highly variable in both severity and distribution, atelectasis can present as a diffuse hazy increase in lung density, patchy areas of lung consolidation, or segmental or lobar collapse (Fig. 25). Atelectasis frequently accompanies other lung disease, such as neonatal pneumonia, and should be suspected as a contributing factor to abnormal lung density and/or aeration, especially when there is rapid change in density patterns.[3]

Surgical Disorders

Pneumothorax with Tension

Occurring most frequently as a complication of mechanical ventilation and secondary interstitial dissection of air, a pneumothorax may progressively increase in size because of a "ball valve" mechanism whereby air enters

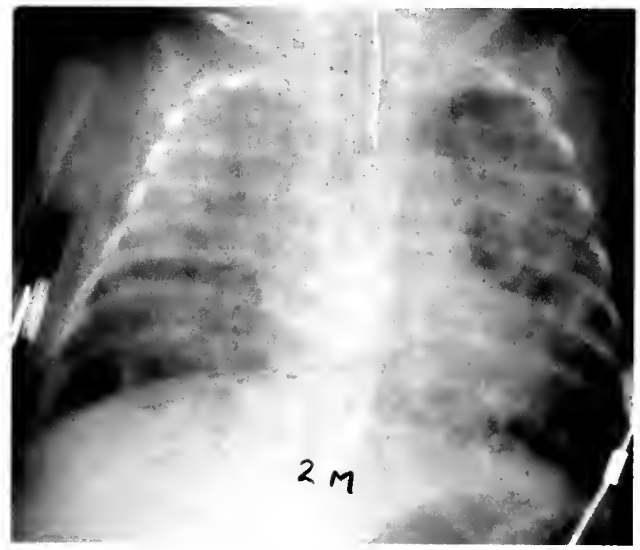

Fig. 15. Two-month-old premature infant with respiratory distress syndrome and on mechanical ventilation with high pressure settings since birth. There are coarse cystic changes throughout both lungs, reflecting severe (Stage IV) bronchopulmonary dysplasia. The endotracheal tube is in good position. (From Reference 3, with permission.)

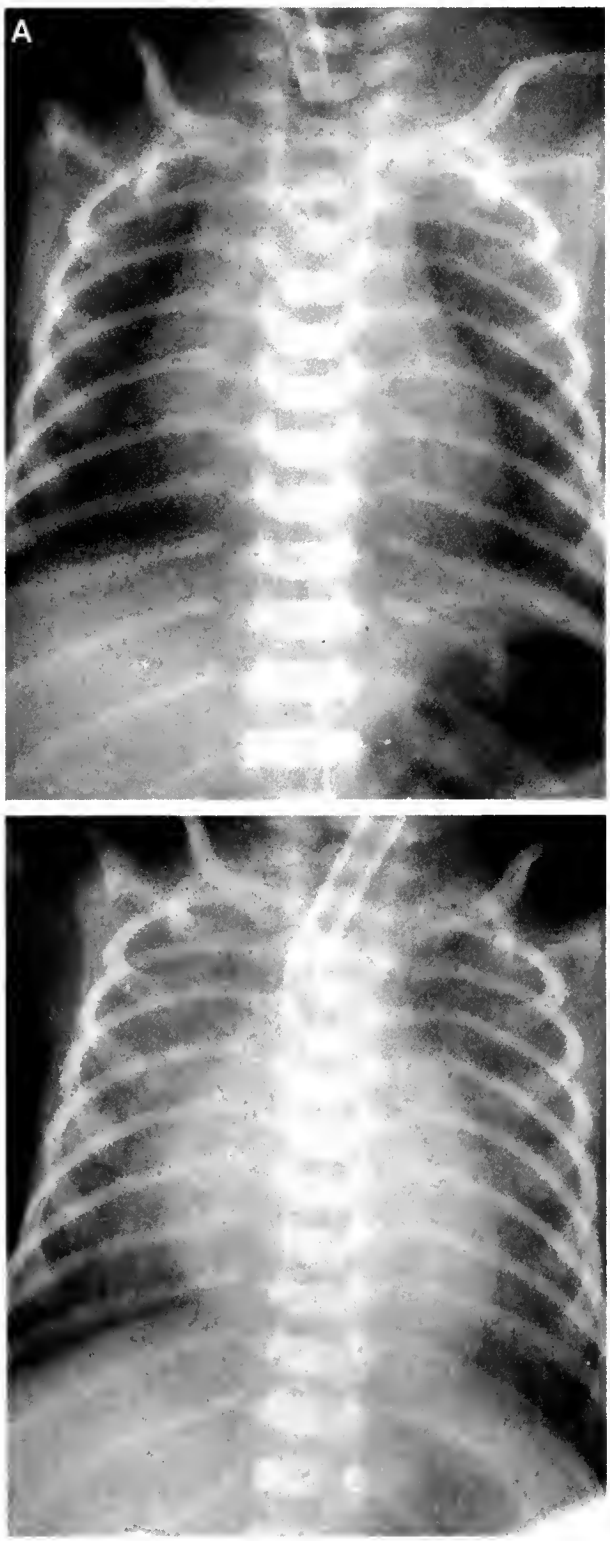

Fig. 16. A: Four-day-old infant with respiratory distress syndrome and mild pulmonary opacification. B: Two days later, in conjunction with increasing distress, bounding pulses, and an audible murmur, the chest radiograph shows an increase in cardiac size and generalized pulmonary density, suggesting a left-to-right shunt via a patent ductus arteriosus, confirmed by echocardiography. The ductus closed with indomethacin. (From Reference 3, with permission.)

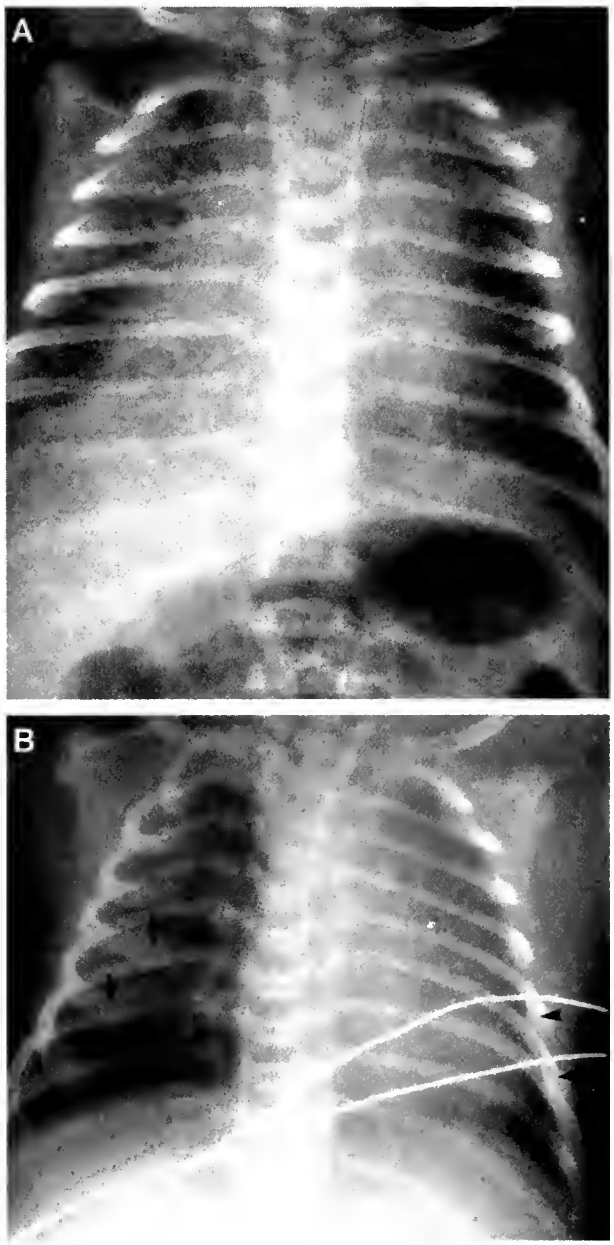

Fig. 17. Transient respiratory distress of the newborn (TRDN). Postnatal respiratory distress in a near-term newborn delivered by cesarean section. A: Initial chest radiograph taken within hours of birth shows a diffuse, bilateral increase in interstitial pulmonary density, with obscuring of the cardiac borders and probably a small right pleural effusion. The position of the clavicles well above the first ribs indicates this to be an apical lordotic projection. B: Repeat chest radiograph the following day shows pronounced clearing of the pulmonary density and a near-normal appearance to the lungs. This rapid an improvement in interstitial pulmonary density strongly suggests retained fetal lung fluid or TRDN as the underlying abnormality. If this pattern of density persists beyond 48 hours of life, TRDN ceases to be a reasonable diagnosis, and neonatal pneumonia or aspiration would become more likely diagnoses. Note the degree of patient left-rotation, as indicated by the anterior ends of the right ribs (arrows) projecting over the right lung field, compared with the anterior ends of the left ribs (arrowheads) projecting farther laterally. (From Reference 3, with permission.)

Fig. 18. Anteroposterior portable chest radiograph in a newborn with severe respiratory distress immediately after birth, requiring intubation and assisted ventilation at high pressure settings. The endotracheal tube (arrow) is in satisfactory position. Both lungs are hyperinflated and show prominent coarsening and accentuation of parenchymal markings, changes highly suggestive of meconium aspiration. Meconium was present in large quantity below the vocal cords. (From Reference 3, with permission.)

the pleural space in excess of leaving.[2,3,7] The resultant increase in intrapleural air pressure appears on the AP supine chest radiograph as an increase in volume and lucency of the involved hemithorax, accompanied by contralateral mediastinal shift, variable collapse of the ipsilateral lung, and depression of the ipsilateral diaphragm (see Figs. 8 and 11).[2,3,7,16]

Congenital Diaphragmatic Hernia

Reflecting herniation of intestinal contents through the posterior pleuroperitoneal foramen, and occurring on the left in 75% of cases, congenital diaphragmatic hernia (CDH) usually causes severe respiratory distress in the newborn.[2,3,7,16,69] Initially, the chest radiographic appearance of CDH may be primarily of an intrathoracic soft tissue mass.[2,3,16] With progression of ingested air through the intestinal tract, the more typical appearance of clustered lucencies with soft tissue septations, reflecting air-containing intestine, is seen radiographically (Fig. 26).[2,3,7,16,69] The classic triad of CDH consists of unilateral intrathoracic intestine, contralateral shift of the mediastinum, and a paucity of intestine in the abdomen, which has a scaphoid appearance on clinical examination.[2,3,7,16,69] Surgical correction of the diaphragmatic defect in conjunction with return of intrathoracic abdominal structures to their proper location is required in those neonates with sufficient duration of survival. Many newborns with CDH require temporary life support via ECMO to control associated pulmonary hypertension.[15]

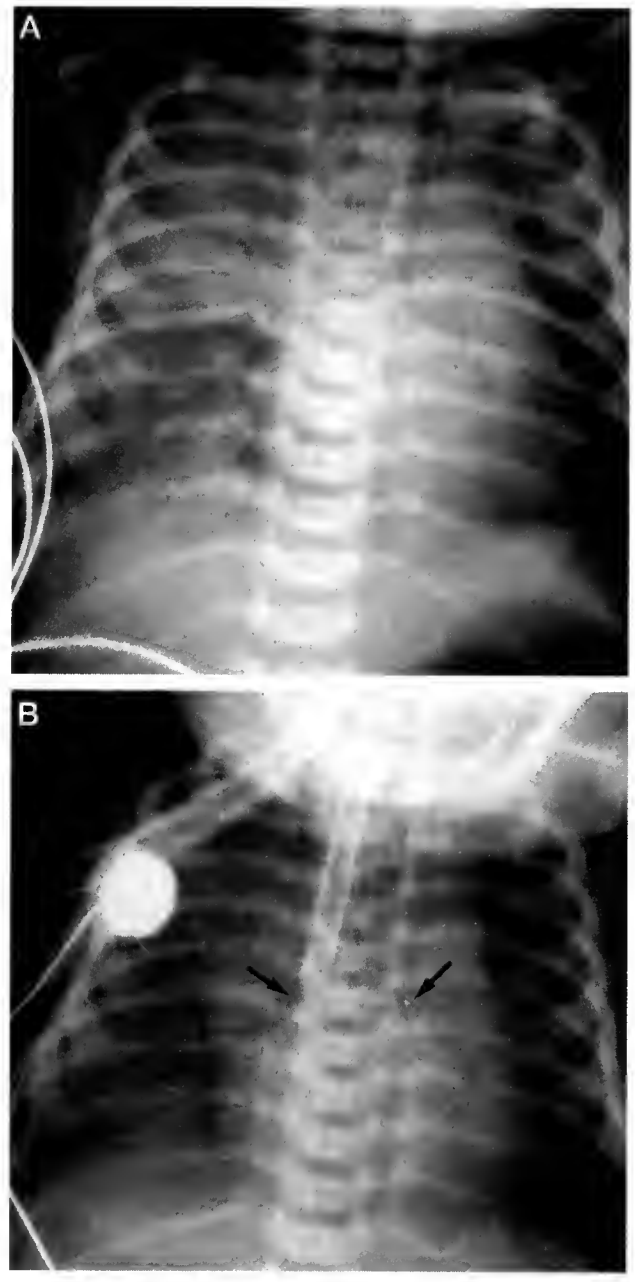

Fig. 19. Newborn with postnatal respiratory distress caused by amniotic fluid aspiration. A: The initial postnatal radiograph shows patchy increase in density, primarily of the right lung. B: Repeat chest radiograph later in the day, and following intubation, shows improvement in lung aeration and decrease in density, but with the tip of the endotracheal tube positioned too low, at the orifice of the right main bronchus. Note the right and left main bronchi (arrows). Although the carina is often not well seen on the anteroposterior chest radiograph, its approximate location can be determined by tracing the main bronchi medially and cephalad to their approximate junction point. (From Reference 3, with permission.)

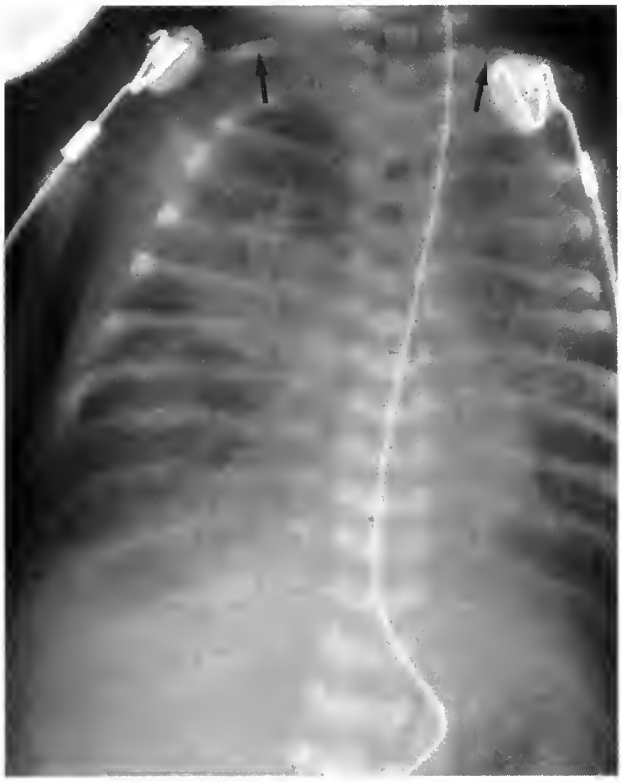

Fig. 20. Neonatal pneumonia. Ten-day-old premature newborn with severe respiratory distress caused by *Pseudomonas* pneumonia. The lungs show diffuse, inhomogeneous increase in opacity, and severe hyperinflation, common findings in neonatal pneumonia. The projection is apical lordotic, as indicated by the clavicles (arrows) projecting above the first ribs and the anterior ends of most of the ribs projecting higher than the posterior rib segments. (From Reference 3, with permission.)

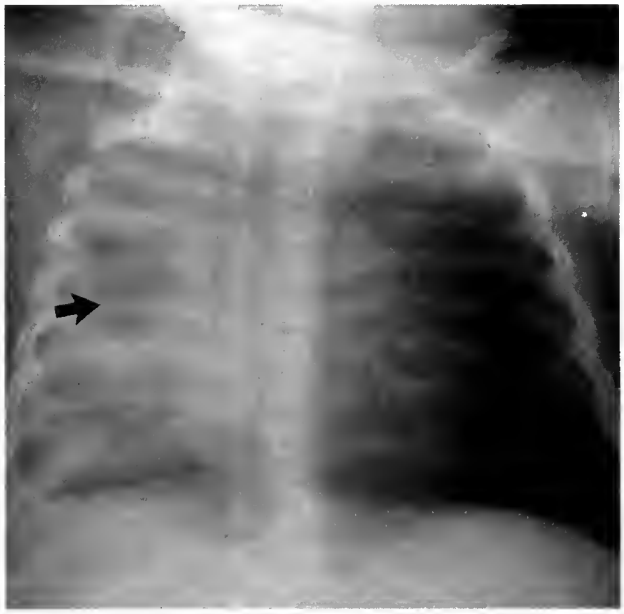

Fig. 21. Staphylococcal pneumonia with pneumatocele formation. Opacification and volume loss of the right lung are accompanied by a large right lung cyst (arrow), reflecting staphylococcal pneumonia with pneumatocele formation. (From Reference 3, with permission.)

Congenital Cystic Adenomatoid Malformation

Reflecting a hamartomatous proliferation of terminal bronchioles into cystic and solid tissue, and with communication of the cystic components with the tracheobronchial tree, congenital cystic adenomatoid malformation (CCAM) has been classified into 3 types.[2] The radiographic appearance of CCAM depends on interval following birth, type of CCAM, and presence of complications such as infection.[2,7,70] Immediately after birth, all types of CCAM appear as a soft tissue thoracic mass with contralateral shift of the heart and other mediastinal structures.[2,7,70] As air replaces the intracystic fluid, a variable pattern of lucency within the cystic component of the CCAM develops radiographically; Type I CCAM will have one or several larger lucent, cystic spaces, while Type III will be predominantly solid soft tissue with variable microcystic lucencies, and Type II will be intermediate in appearance (Fig. 27).[2,7,70] The presence of a normal quantity and distribution of air-containing intestine within the abdomen permits differentiation of CCAM from CDH radiographically.[2,3,7,16,70]

Congenital Lobar Emphysema

Usually limited to 1 or, at the most, 2 lobes, and most likely reflecting bronchial obstruction by a ball valve mechanism, congenital lobar emphysema usually results in respiratory distress of some degree in the neonatal period.[2,3,7,16,71] Fluid trapped in the hyperexpanded lobe early in the postnatal period results in a unilateral opaque mass-like appearance on the chest radiograph.[2,3,7,16,71] With progressive replacement of fluid with air in the ensuing days, a hyperlucent emphysematous-appearing lobe, most commonly the left upper lobe, is the typical radiographic finding (Fig. 28).[2,7,16,71] Because conservative therapy is associated with a high mortality rate, surgical resection of the involved lobe is usually required.[6]

Imaging Abnormalities in the Pediatric Intensive Care Unit

While the most common projection for imaging the chest in a non-neonate child in the ICU is an AP view with the patient lying supine, there is a greater likelihood that a PICU child will be in the semi-erect or upright position. An inclinometer can be attached to the film cassette to indicate the degree of erectness of patient positioning.[72]

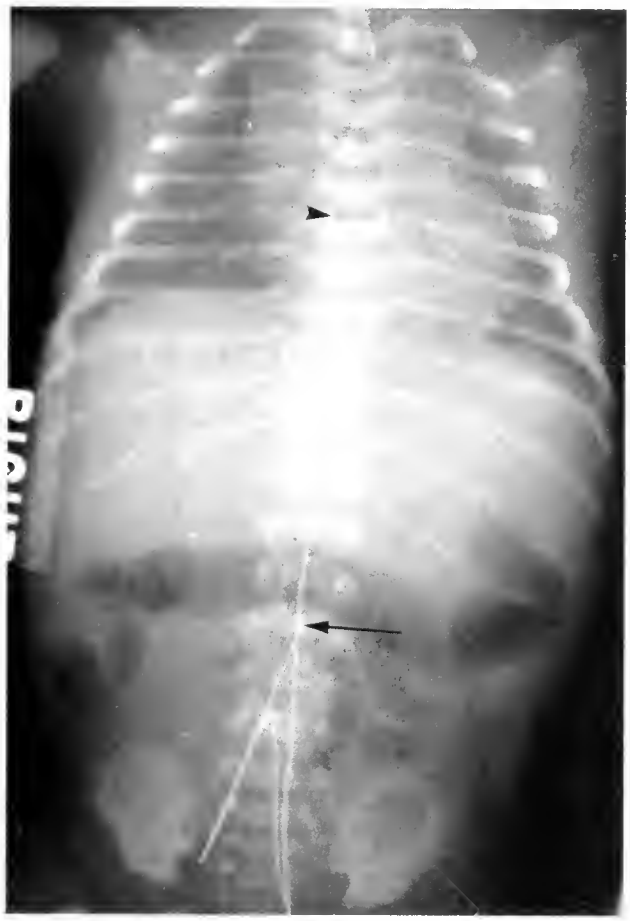

Fig. 22. Premature newborn with respiratory distress. Diffuse pulmonary granularity with air bronchograms suggests respiratory distress syndrome (RDS), but in this patient reflected B-streptococcal pneumonia, which can have a radiographic appearance identical to RDS. Premature rupture of the maternal membranes had occurred. The endotracheal tube is in the intrathoracic trachea, in satisfactory position. The tip of the umbilical arterial catheter (UAC) (arrow) overlies L-3, with preferred UAC catheter tip position being between L-3 and L-5. The umbilical venous catheter (UVC) tip (arrowhead) is intracardiac, higher than the preferred location near the junction of the right atrium and inferior vena cava. (From Reference 3, with permission.)

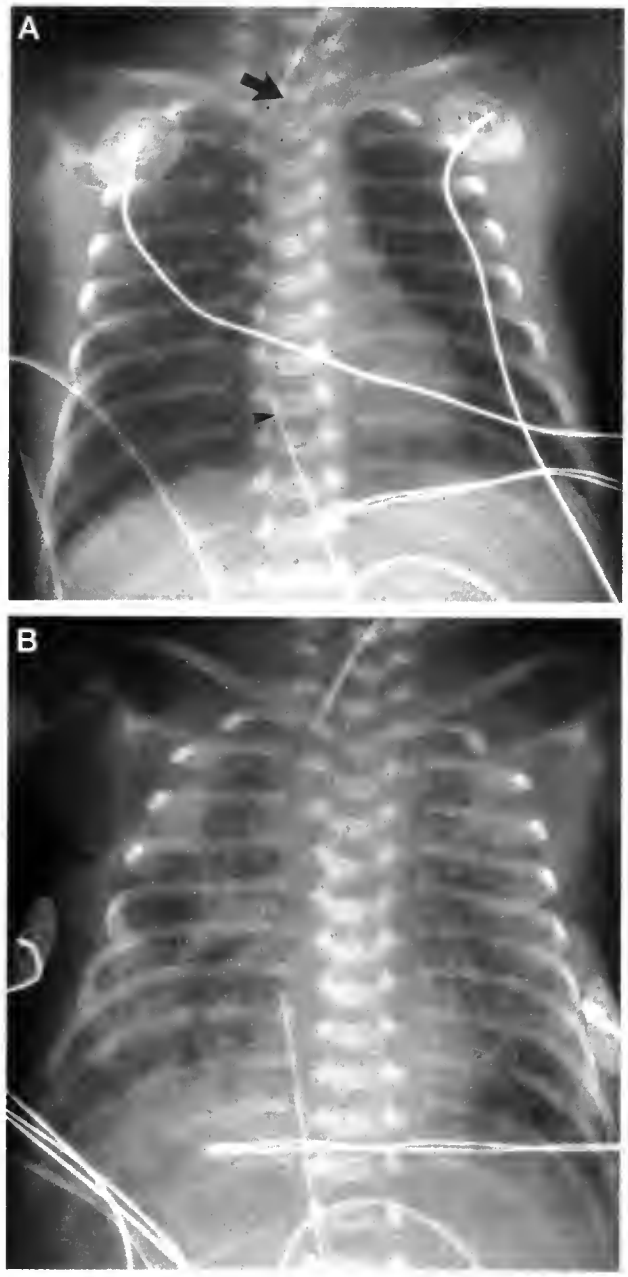

Fig. 23. A: One-day-old infant with significant respiratory distress caused by pulmonary hypertension, who required mechanical ventilation. The lungs are clear, heart size is normal, and the endotracheal tube (ETT) (arrow) is in satisfactory position. The umbilical venous catheter tip (arrowhead) is intracardiac, slightly higher than preferred. B: The following day, a diffuse increase in pulmonary opacification developed bilaterally in conjunction with increasing respiratory distress and blood emanating from the ETT. Transient but massive pulmonary hemorrhage was the cause of the rapid increase in pulmonary opacification, which in many cases is complete, with absence of aeration of the lungs. (From Reference 3, with permission.)

The presence of an air-fluid level in the stomach indicates the patient is in the erect position.[72]

To improve radiographic exposure on a consistent basis, the technologist should either follow a generic "portable technique chart" for exposure factors or maintain an individual patient technique record for successful exposures.[72] A constant kilovoltage should be employed; milliamperage is varied according to patient size and the thickness of the thorax.[72]

The AP projection and shortened tube-to-film distance required with portable chest radiography results in greater magnification of more anteriorly placed structures such as the heart.[72] If the exposure is obtained in relative expiration, the degree of magnification is even greater.[72] Compared to the standard erect posteroanterior chest radiograph with satisfactory inspiration, a supine expiratory AP portable chest radiograph in an adult will increase the

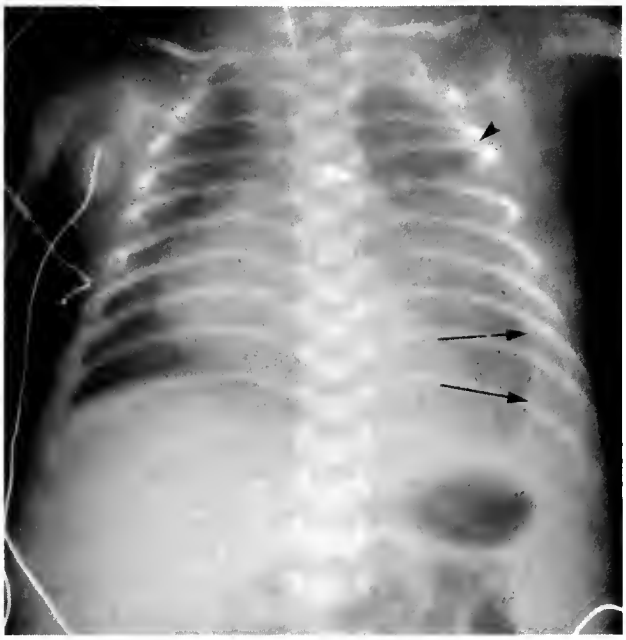

Fig. 24. Left chylothorax following ligation of a patent ductus arteriosus (PDA). One-month-old with mediastinal surgical clip placed for ligation of a PDA. Pleural fluid (arrows) has accumulated in the left pleural space postoperatively, identified as a chylothorax following chest tube drainage, and believed to reflect disruption of the thoracic duct at thoracotomy. Localized left upper rib deformity identifies the thoracotomy site (arrowhead).

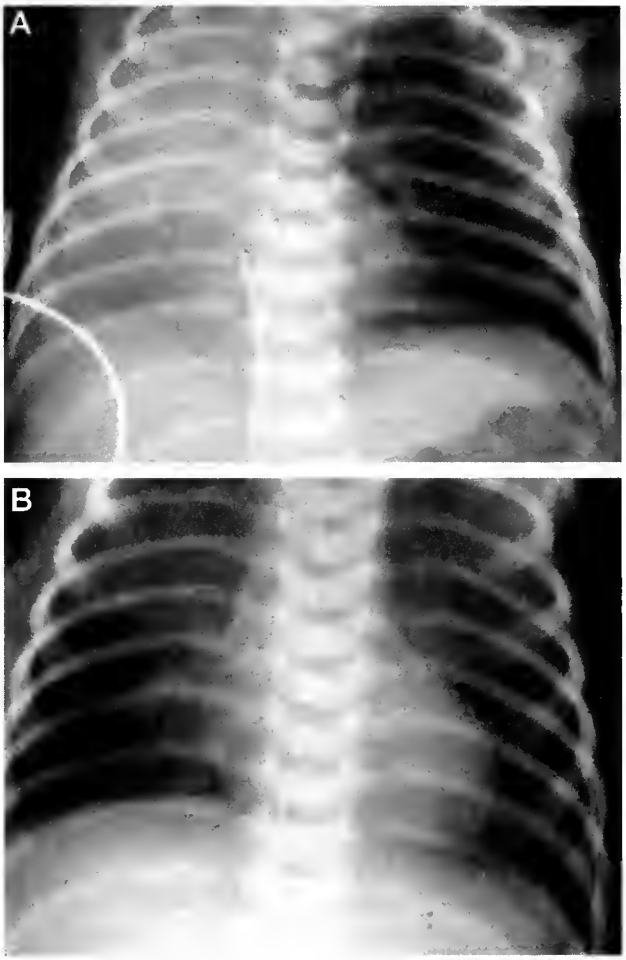

Fig. 25. A: Morning radiograph shows generalized atelectasis of the right lung, with right-shifted mediastinum. B: Repeat chest radiograph that evening shows complete resolution of the right-sided atelectasis, with return of normal aeration. Rapid development and resolution of pulmonary densities in the neonate strongly suggest atelectasis. (From Reference 3, with permission.)

apparent transverse cardiac diameter by as much as 11%, the cardiothoracic ratio by 25%, and the apparent width of the upper mediastinum by about 50%.[72]

Tubes, Lines, and Catheters

Most children in the PICU require ventilatory support for at least part of their stay.[2] Initial mechanical ventilatory support is almost always with an ETT, which is at least partially opaque, permitting visualization on the chest radiograph. The tip of the ETT should be within the intrathoracic portion of the trachea, preferably half way between the thoracic inlet and the carina, and projecting just below the inferomedial ends of the clavicles.[2] Descent of the ETT is associated with flexion of the neck, while ascent occurs with neck extension.[73] Endotracheal tubes with metallic reinforcement may have a radiolucent distal segment that is not visible on the chest radiograph, resulting in potential misjudgement of ETT tip position.[73] As with neonates, the ETT in the pediatric patient can be malpositioned within the trachea as well as in the esophagus.[73] Tracheal or laryngeal laceration is an uncommon but potentially serious complication of endotracheal intubation.[73]

Children requiring long-term mechanical ventilation or suctioning will usually undergo tracheostomy tube placement.[73] Usually inserted through a stoma at the level of the third tracheal cartilage, the tracheostomy tube tip should be 1–2 cm above the carina, depending on patient size.[73]

A central venous pressure (CVP) catheter is often utilized in PICU patients to monitor CVP and administer fluids.[73] These catheters are of sufficient size and opacity to be visible on the chest radiograph. The subclavian, internal jugular, and femoral veins are the most common access sites.[73] The preferred location for the tip of the CVP catheter is in the superior vena cava.[2,73] Catheter malposition and pneumothorax are the most common complications of CVP catheter placement that can be identified on the chest radiograph.[73]

Catheters designed for long-term venous access are increasingly utilized in oncology, bone marrow transplant, and solid organ transplant patients.[73] Usually placed surgically via the subclavian venous route, the Hickman, In-

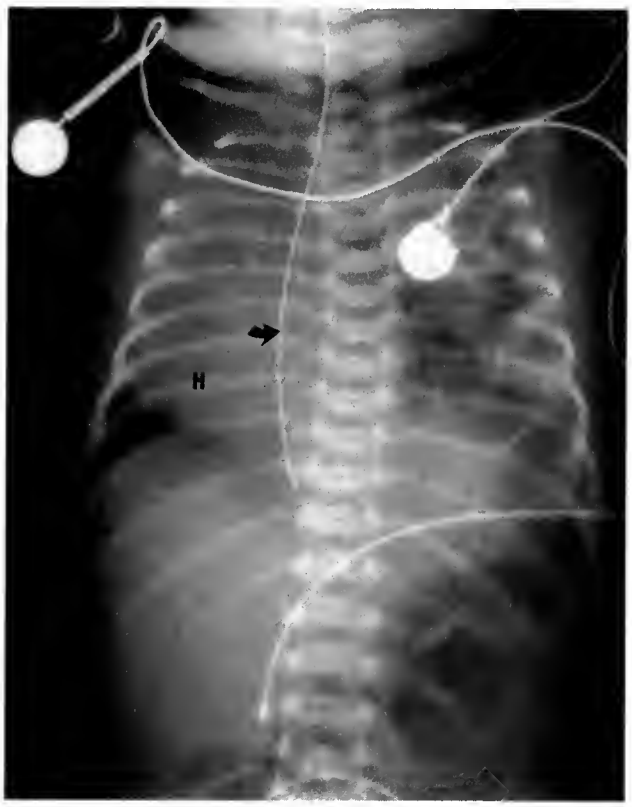

Fig. 26. Congenital diaphragmatic hernia. Newborn with immediate postnatal respiratory distress. Anteroposterior radiograph of the chest and upper abdomen shows a bubbly, cystic appearance to the left hemithorax, reflecting intrathoracic bowel. The heart (H) is displaced to the right. The position of the nasogastric tube (arrow) indicates rightward shift of the esophagus as well. Note the continuity of bowel extending from the left upper quadrant into the left hemithorax. With this condition, the abdomen will frequently have a scaphoid appearance because of the absence of intraabdominal bowel. (From Reference 3, with permission.)

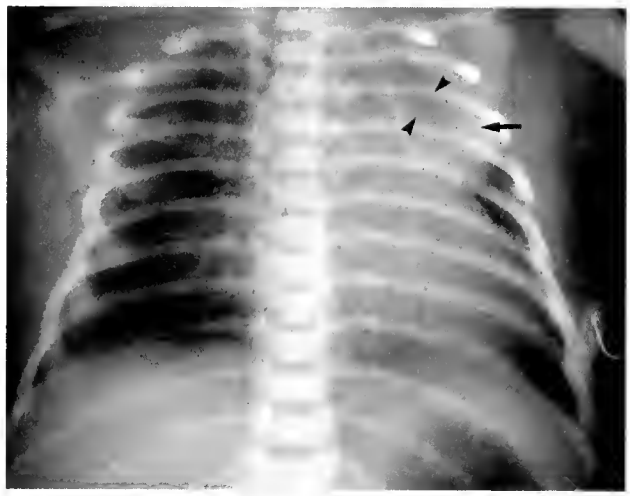

Fig. 27. Congenital cystic adenomatoid malformation. Newborn with mild respiratory distress and on mechanical ventilation. Antenatal ultrasonography had suggested a left-sided chest mass. The anteroposterior chest radiograph shows an increase in soft tissue density occupying the upper third of the left hemithorax (arrow) with a central area of lucency (arrowheads). A congenital cystic adenomatoid malformation was found at surgery. (From Reference 3, with permission.)

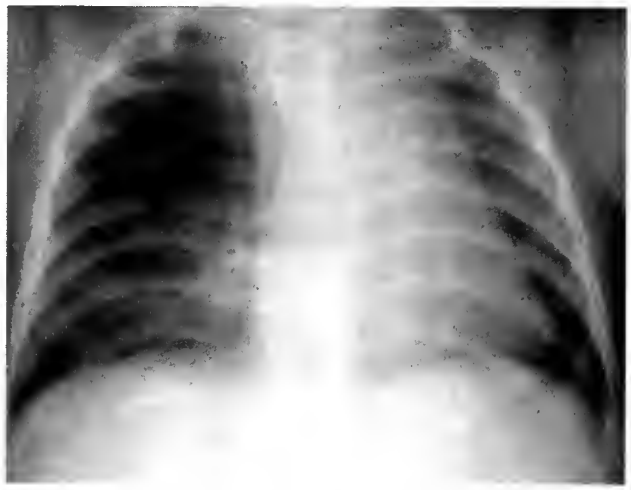

Fig. 28. Congenital lobar emphysema. There is relative hyperlucency and hyperinflation of the upper portion of the right lung in conjunction with partial compressive atelectasis of the right lower lobe. Congenital lobar emphysema involving the right upper lobe was found at surgery. (From Reference 3, with permission.)

fusaport, and Port-a-cath systems are sufficiently opaque to be visible on the chest radiograph.[73]

Transvenous pacemakers and transcutaneous pacing wires are infrequently utilized in pediatric patients, almost always in the clinical setting of cardiac arrhythmias or congenital cardiac disease. A variety of cardiac reconstructive or palliative stents and conduits are used in the surgical correction of congenital cardiac disease. Not all of these devices are of sufficient opacity to be detectable on the chest radiograph.

Atelectasis

Usually due to hypoventilation from central depression, general anesthesia, or splinting from trauma or thoracic or abdominal surgery, atelectasis is the most frequent chest radiographic abnormality in the PICU.[74] The radiographic findings in atelectasis are highly variable, from that of clear lungs of mildly diminished volume to total consolidation and volume loss of a lobe or entire lung.[74,75] Linear densities in the lung, especially at the lung bases, mediastinal shift toward the increased lung opacity, and a rapidly changing pattern of pulmonary density suggest atelectasis.[74,75] Because the left lower lobe is hidden behind the density of the heart on the AP chest radiograph, it is the area most frequently missed in examining for atelectasis.[74]

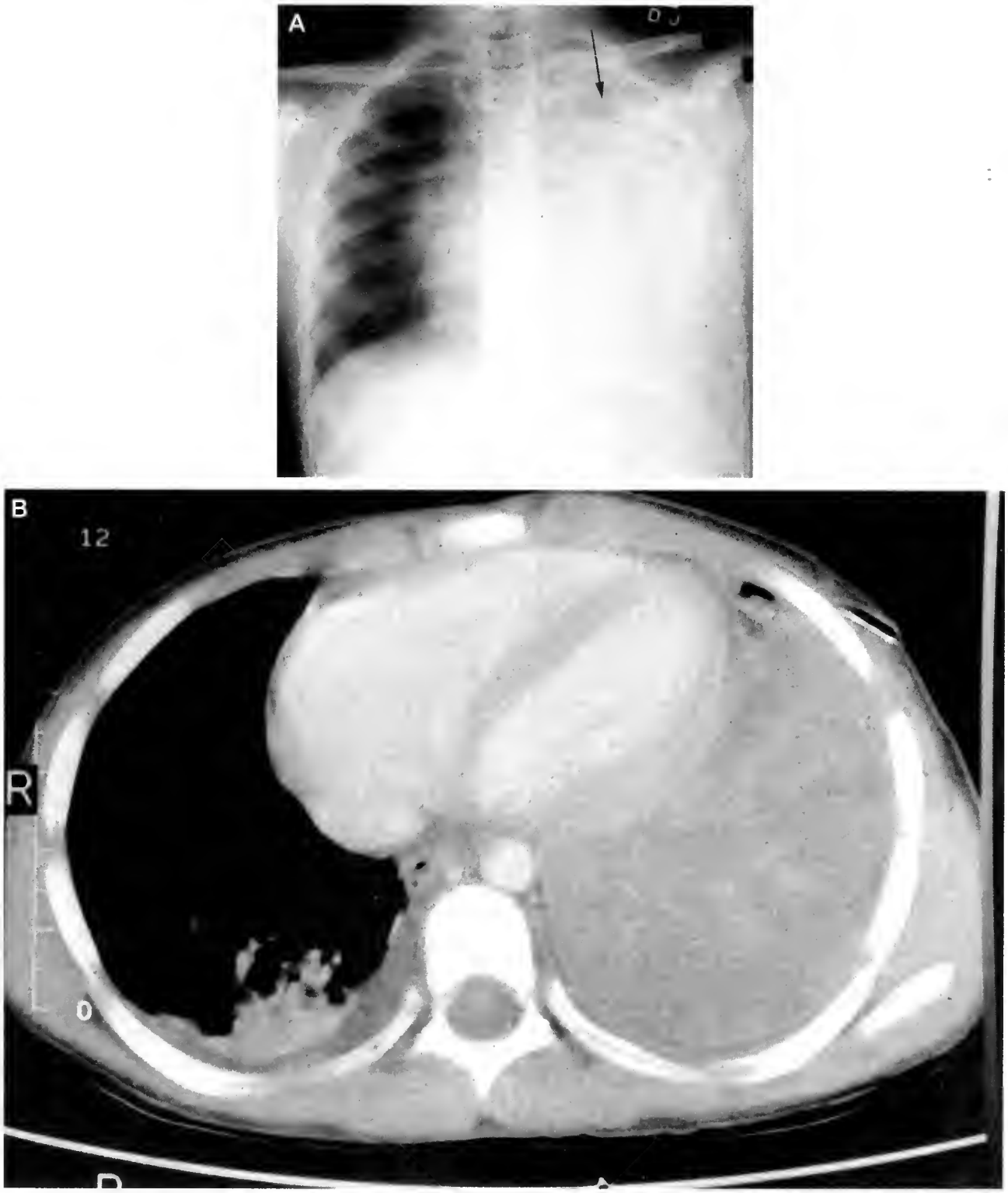

Fig. 29. Ten-year-old male with pneumonia. A: Anteroposterior portable erect chest radiograph shows near-complete opacification of the left hemithorax, with minimal residual aeration in the left upper lobe (arrow), and suggestion of mild contralateral mediastinal shift. This exam did not determine how much of the left-sided opacification was due to consolidation from atelectasis and/or pneumonia and how much from associated pleural fluid. B: Contrast-enhanced computed tomography (CT) image through the lower thorax shows inhomogeneous opacification of the entire left lower lobe, due to consolidative pneumonia, with very little associated left pleural fluid. There is patchy consolidation with limited pleural fluid on the right. More cephalad CT views showed partial left upper lobe and lingular consolidation.

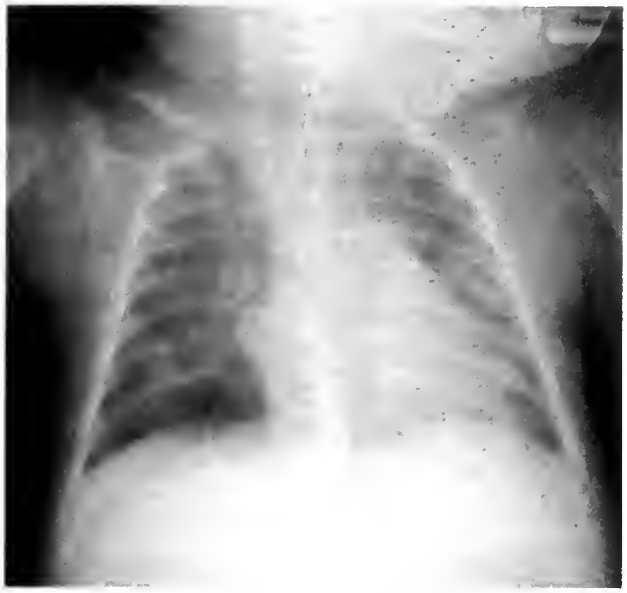

Fig. 30. Respiratory syncytial virus (RSV) pneumonia. 10-week-old infant with increasing respiratory distress. Anteroposterior portable chest radiograph in the neonatal intensive care unit shows areas of patchy alveolar and irregular interstitial density that was confirmed as representing RSV pneumonia. The patient fully recovered.

Pneumonia and Complications

Pneumonia in PICU patients may be either community-acquired (resulting in patient admission to the PICU) or nosocomial (occurring more than 72 hours after admission).[74] The former are more likely to be caused by viruses, *Mycoplasma* organisms, or Gram-positive cocci, the latter by Gram-negative bacteria.[74] The radiographic findings in pneumonia are highly variable, ranging from a near-normal appearance of the lungs, to focal or diffuse interstitial densities, to focal or diffuse consolidation of lung parenchyma (Fig. 29).[74,76–78] Atelectasis frequently accompanies pneumonia and complicates the interpretation of chest radiographic findings.[78] In general, a pneumonia of viral or mycoplasma origin is more likely to produce an interstitial change (Fig. 30), while a bacterial pneumonia would produce patchy or diffuse areas of consolidation.[74–76,78,79] Lobar consolidation most commonly reflects *Streptococcus pneumoniae* or *Haemophilus influenzae* infection (Fig. 31).[75]

Pleural effusion, empyema, bronchopleural fistula, and lung abscess are potential complications of pneumonia.[74] Pleural effusion accompanying pneumonia suggests a pneumococcal, staphylococcal, tuberculous, or *Haemophilus* etiology.[2] Both pleural effusions and empyema appear on the chest radiograph as zones of soft tissue density displacing aerated lung from the inner thoracic wall margin, and cannot be reliably differentiated on the chest radiograph alone (see Fig. 31). A collection of air and fluid in the pleural space would suggest either a bronchopleural fistula and/or an empyema (pyopneumothorax) (Fig. 32).[74] *Staphylococcus aureus* is the likely cause of pyopneumothorax.[2,75] A lung abscess, most likely reflecting staphylococcal or *Haemophilus influenzae* infection, would appear as a lung parenchymal, not pleural-based, density that can range from solidly soft tissue to a thick, irregular rind with central air collections.[2,75,80] As viewed on an erect chest radiograph, an air-fluid level occurring with a lung abscess would be confined to the area of parenchymal opacity, not cross fissures, and would not extend to the chest wall, while an air-fluid level in a hydropneumothorax, hemopneumothorax, or pyopneumothorax would have a longer air-fluid interface and extend to the chest wall.[78] Thoracic CT provides significant additional diagnostic information relative to both lung involvement in pneumonia and evaluation of the above potential complications, but may not be applicable to the PICU patient because of the lack of portability for bedside evaluation. Ultrasound of the thorax and pleural spaces can provide differentiation of simple (effusions) from complex (empyema) pleural fluid collections, and can assist in localizing fluid for diagnostic or therapeutic thoracentesis.[75] A pneumatocele is an air-filled cyst, usually thin-walled and devoid of fluid, that can result from staphylococcal or Gram-negative lung infection.[2,75]

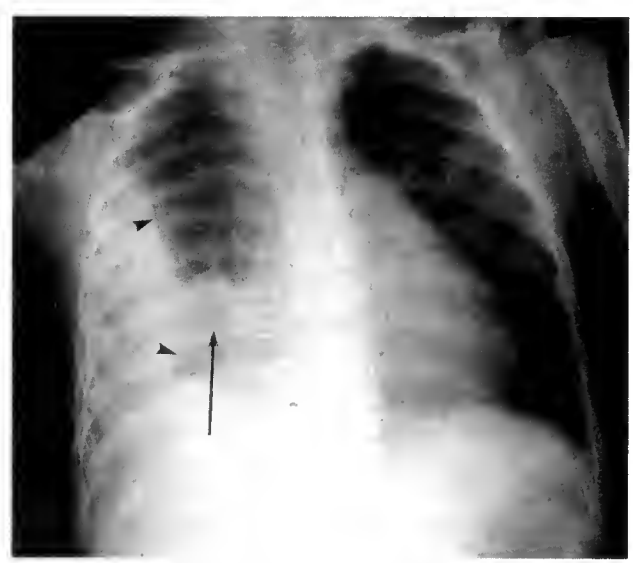

Fig. 31. Pneumonia with empyema. 21-month-old male with pneumonia and with lack of clinical improvement despite appropriate therapy. Portable chest radiograph shows consolidation in the right lower lobe due to pneumonia (arrow) and accompanied by fluid in the adjacent pleural space (arrowheads) (needle aspiration revealed empyema).

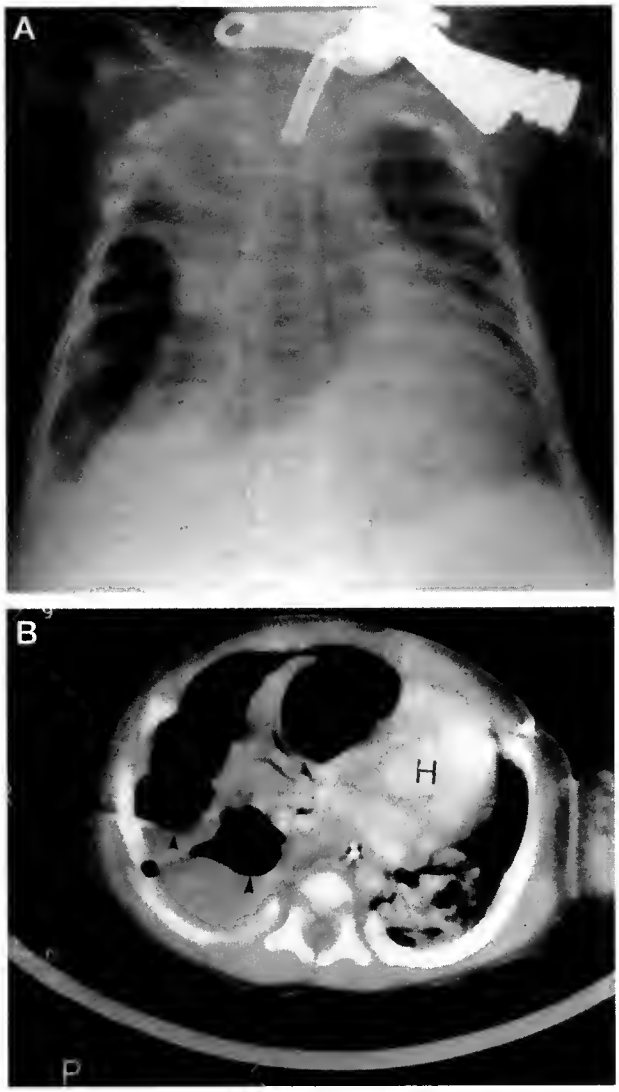

Fig. 32. Five-month-old infant (who had been born prematurely) with bronchopulmonary dysplasia (BPD) and ventilator dependence. Recent previous chest radiographs had showed a right-sided infiltrate identified as staphylococcal pneumonia. A: Radiograph shows a complex appearance to the right hemithorax, consisting of multiple areas of loculated air and soft tissue, and mass effect producing contralateral mediastinal shift. Appearances suggest a pyopneumothorax. B: Contrast-enhanced computed tomography image through the lower thorax shows complex air and soft tissue changes with air-fluid levels (arrowheads) in the right hemithorax, again with left-shift of the heart (H) and other mediastinal structures. A staphylococcal pyopneumothorax was confirmed on catheter drainage of the right pleural space.

Pulmonary Infection in the Immunocompromised Child

Causes of immunocompromise in the pediatric population include congenital immunodeficiency disorders, acquired infection (especially AIDS), and immunosuppression due to chemotherapeutic agents.[2] Although a variety of etiologic agents can cause pulmonary infection in the immunocompromised child, invasive aspergillosis, tuberculosis (TB), and especially *Pneumocystis carinii* are the most common.[2,81] Variable pulmonary opacification, with or without cavitation, adenopathy, and/or calcification, are potential chest radiographic findings with invasive aspergillosis.[2,82] Initial pulmonary infection with TB produces localized airspace disease in any pulmonary segment or lobe, regional lymph node enlargement and/or pleural effusions.[2] Vascular seeding of tuberculous organisms to the lungs may result in miliary TB, which appears as multiple, small (2–3 mm) nodular opacities scattered throughout both lungs.[2] *P. carinii* pneumonia produces a nonspecific interstitial pattern initially, with progression to diffuse airspace disease having a fine "ground-glass" appearance.[2,81]

Aspiration Pneumonitis

Most likely to occur in the neurologically impaired or intubated child, aspiration pneumonitis can result from the introduction into the airways and lungs of both toxic (eg, acidic gastric juices) and bland (eg, water, barium) fluids or solids, or infected secretions.[74] Aspiration of gastric contents can produce a severe chemical pneumonitis, resulting in diffuse pulmonary opacification suggesting pulmonary edema.[75] The initial radiographic findings may be interstitial, airspace, or mixed, and may be predominantly perihilar or basilar in distribution (Fig. 33).[74]

Acute Smoke Inhalation

The chest radiographic findings in acute smoke inhalation can vary from normal to interstitial or alveolar densities suggesting edema.[74,75] Subglottic edema, thickening of the trachea and bronchi, and perihilar interstitial changes are considered early chest radiographic signs of significant lung injury related to burns and smoke inhalation.[74] Diffuse air trapping and pulmonary hyperinflation associated with bronchiolar damage may be the presenting radiographic findings.[75] Excess crystalloid fluid administration coupled with low colloid oncotic pressure from surface protein loss can contribute to pulmonary interstitial edema.[74]

Near-Drowning

In near-drowning, the chest radiographic findings vary from normal to mild interstitial changes in milder cases, to diffuse alveolar edema in more severe cases (Fig. 34).[2,74,83] The composition of the intrapulmonary fluid (salt water vs fresh water) has little effect on the chest radiographic findings.[2,74,83]

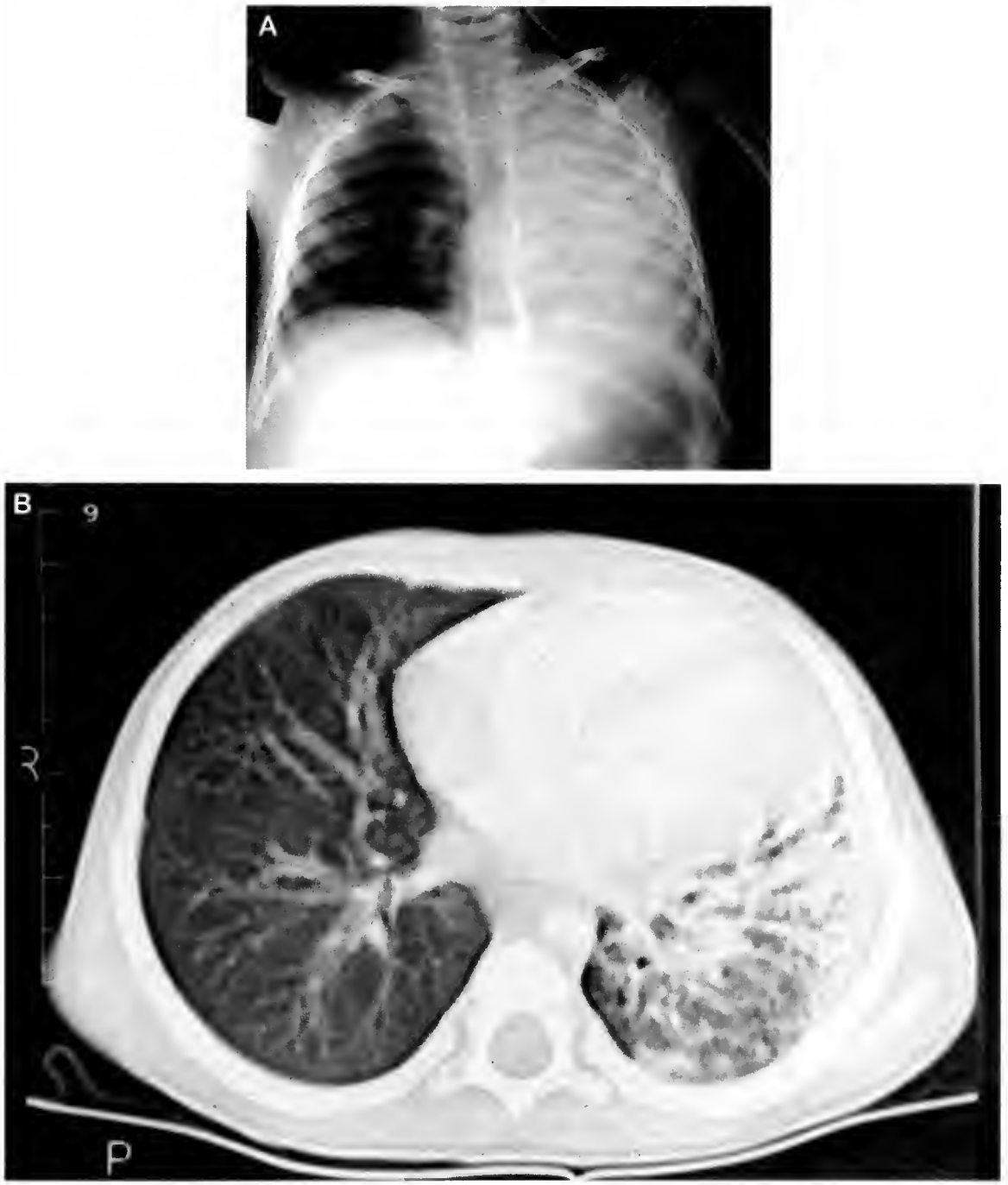

Fig. 33. Pneumonia secondary to foreign body aspiration. 23-month-old with onset of cough and fever. A: Anteroposterior portable chest radiograph shows extensive consolidation throughout the left lung, accompanied by multifocal areas of limited lung aeration. B: Contrast-enhanced computed tomography image through the lower chest shows extensive nonhomogeneous left lower lobe consolidation with limited volume loss. Because of a lack of appropriate response to treatment, bronchoscopy was performed, revealing aspirated peanut fragments in the left lower lobe bronchus.

Hydrocarbon Aspiration and Pneumonitis

Aspiration by children of hydrocarbons such as kerosene, gasoline, or furniture polish may induce a chemical pneumonitis within hours.[2,84] The resultant surfactant destruction, bronchial and bronchiolar necrosis, and small vessel thromboses may result in alveolar atelectasis, hypoxemia, and bronchopneumonia.[2,84] Usually not recognizable for 6–12 hours after aspiration, the primary chest radiographic finding with hydrocarbon pneumonitis is

Fig. 34. Near-drowning. 8-year-old male found submerged in a swimming pool. Anteroposterior portable supine chest radiograph in the pediatric intensive care unit shows diffuse bilateral pulmonary airspace opacification. The cardiac silhouette is mildly prominent. The endotracheal tube and other support lines are satisfactorily positioned.

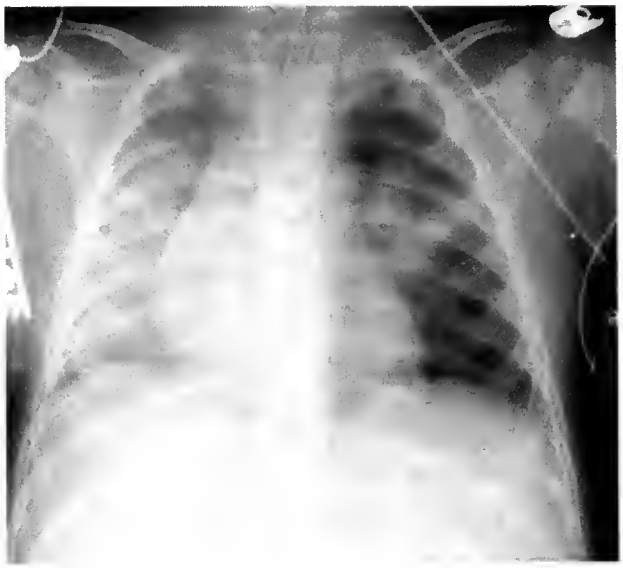

Fig. 35. Chemical pneumonitis. 14-month-old infant who ingested kerosene while unattended. Increasing respiratory distress required admission to the pediatric intensive care unit and intubation for mechanical ventilatory assistance. The anteroposterior portable supine chest radiograph shows bilateral but asymmetrical (worse on the right) alveolar infiltrates compatible with chemical pneumonitis. There is no associated pleural effusion.

fluffy, confluent opacities that tend to persist for weeks or months, lagging behind clinical improvement (Fig. 35).[2] Pneumatocele formation may occur within areas of pneumonia and necrosis.[2]

Cardiac Failure and Pulmonary Edema

Pulmonary edema reflects the accumulation of excess fluid in the interstitium of the lung initially, followed by the alveolar spaces.[75,85] Pulmonary edema can result from left-sided cardiac failure or from noncardiac causes such as head trauma or drug reaction.[75,85] In general, the chest radiographic findings in pulmonary edema evolve from an increased prominence of the interstitial lung markings initially, to the development of patchy or diffuse confluent lung opacification that tends to predominate in the perihilar regions (Fig. 36).[2,75,85] Pulmonary edema due to cardiac failure (compared to noncardiac etiologies) is more likely to be associated with enlargement of the cardiac silhouette, Kerley lines, peribronchial cuffing, and pleural effusions.[85]

Patient positioning influences the radiographic appearance of pulmonary edema. With the patient in the erect position, a redistribution of blood flow from predominantly lower lobes to upper lobes can be an early sign of developing edema.[85] Reliability of measurement of the cardiothoracic ratio as an indication of cardiomegaly is greatest

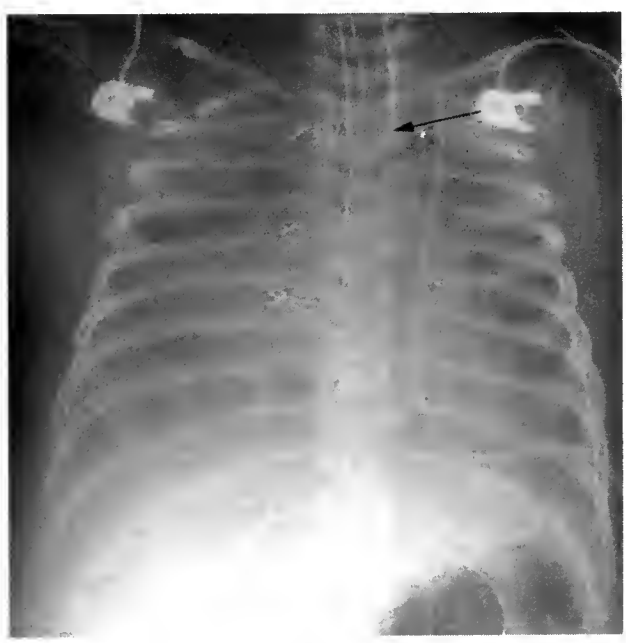

Fig. 36. Anteroposterior supine portable chest radiograph shows extensive bilateral pulmonary opacification due to congestive cardiac failure. The left pleural catheter was placed for control of a pleural effusion. Endotracheal, nasoenteric, and left internal jugular venous (arrow) catheter and tube position is satisfactory.

with the patient erect.[85] The AP supine chest radiograph frequently mandated by the patient's clinical status will produce a redistribution of blood flow from lower to upper

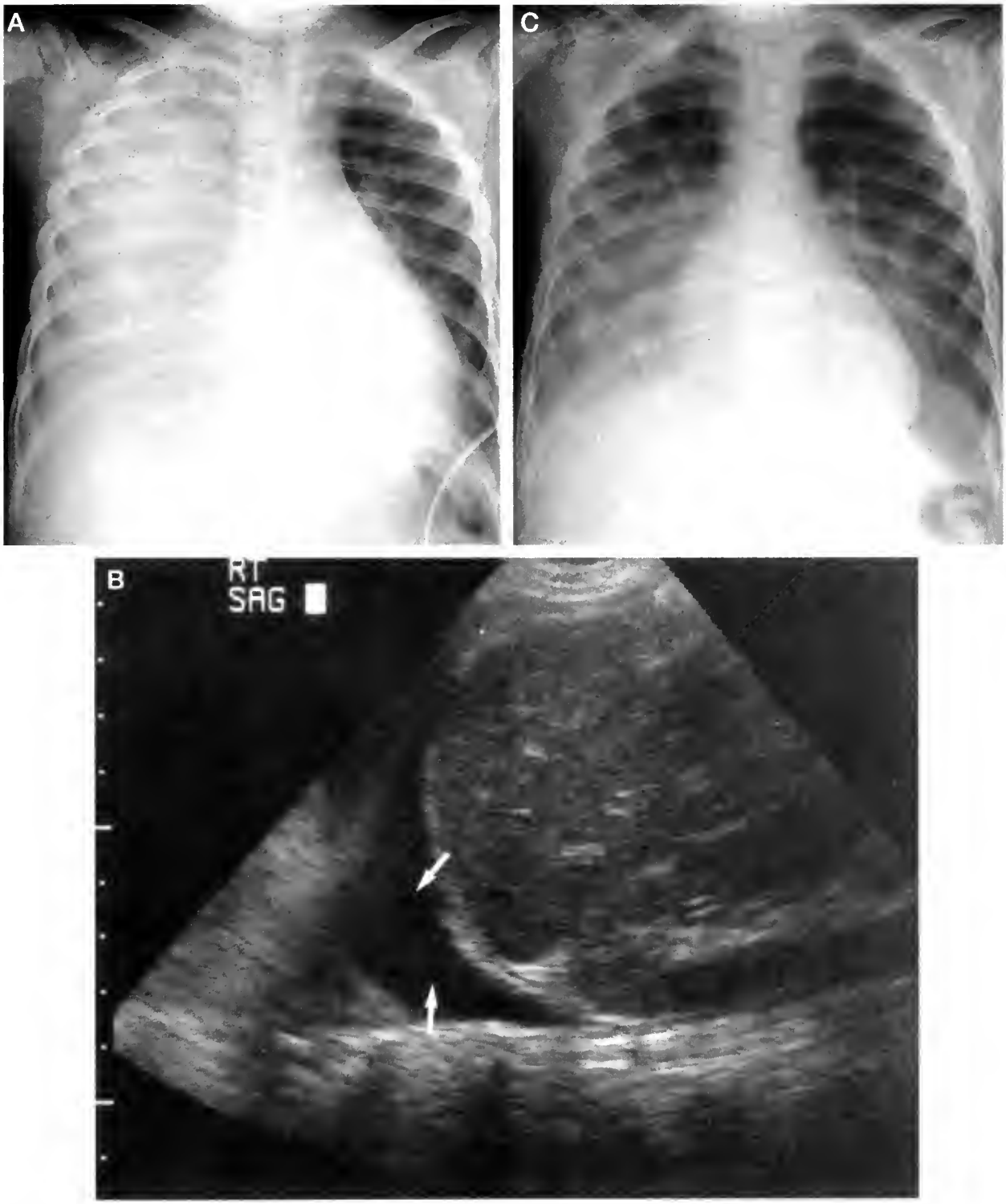

Fig. 37. Acute chest syndrome in sickle cell disease. 3-year-old with sickle cell disease and acute onset of chest pain, fever, and cough. A: Anteroposterior portable chest radiograph shows almost complete opacification of the right hemithorax, with mild contralateral shift of mediastinal structures, findings compatible with extensive lung consolidation with pleural fluid. B: Sagittal ultrasound image of the right lower chest and upper abdomen shows a large quantity of primarily subpulmonic pleural effusion (arrows). C: Anteroposterior portable chest radiograph after right pleural chest tube placement and fluid drainage shows improved aeration of the right lung but persisting bilateral lower lobes infiltrates. Mild enlargement of the cardiac silhouette is better seen after pleural fluid removal.

Fig. 38. Sickle cell crisis. 16-year-old female with sickle cell anemia. Rapidly increasing respiratory distress associated with fever and coughing resulted in admission to the pediatric intensive care unit. Anteroposterior portable supine chest radiograph shows enlargement of the cardiac silhouette, prominent pulmonary vasculature (suggesting high output cardiac failure), and bilateral lower lobe infiltrates (suggesting pneumonia).

lobes, as well as an increase in apparent heart size, complicating the interpretation of the chest radiograph as an indicator of cardiac failure and pulmonary edema.[85]

Acute Chest Syndrome in Sickle Cell Disease

The acute onset of chest pain, fever, and hypoxia, termed the acute chest syndrome, can occur in children with sickle cell anemia.[2,86] Transient pulmonary opacities that are usually bilateral, irregular, and indistinct, and accompanied by variable cardiomegaly, will typically occur on the chest radiograph (Figs. 37 and 38).[2,86]

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS), or "shock lung," represents acute lung injury secondary to a variety of insults that result in profound hypoxia or hypotension, and which may include aspiration, drug reactions, hematologic disorders, infection, and trauma.[2,75,87–90] Although there is considerable variability in findings, the chest radiograph is usually normal for the first 12–24 hours.[2,85,89,90] Abnormal radiographic findings during the initial 12 hours usually reflect a complication such as aspiration pneumonia, fluid overload, or congestive cardiac failure.[75,89,90] The more fulminant the lung injury, the shorter the latent period before onset of radiographic findings.[75,89,90]

A perihilar haze with linear opacities extending from the hilum consistent with interstitial edema, is the initial radiographic abnormality.[75,85,89,90] Linear densities coalesce into patchy areas and then more generalized areas of lung opacification, often accompanied by reduced overall lung volume because of diminished lung compliance.[75,85,89,90] Cardiomegaly is typically absent.[85] PIE may accompany ARDS.[91] Rapid progression of disease, from localized confluent opacities to diffuse airspace opacification within 24 hours, is common (Fig. 39).[2,85,89,90] With successful treatment and lung recovery, the lungs may return to a normal appearance within days to weeks, or show focal areas of scarring consistent with fibrosis.[85,89,90]

Abnormal Pulmonary and Pleural Air and Fluid Collections

Pulmonary Interstitial Emphysema. Occurring almost exclusively in children on mechanical ventilatory assistance, PIE presents radiographically as lucent streaks radiating in a disorganized fashion from the hila to the lung periphery.[91] Unlike air bronchograms, these lucencies do not branch or decrease in caliber as they progress toward pleural surfaces.[91]

Pneumatoceles (Air Cysts). Localized accumulation of air in the lung interstitium can result in the formation of air

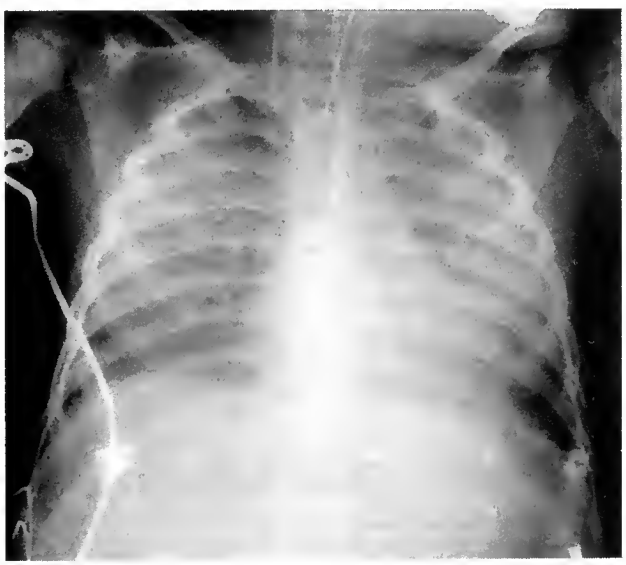

Fig. 39. Acute respiratory distress syndrome (ARDS). 4-year-old male with recurrent seizures who developed aspiration pneumonia followed by progressive deterioration of respiration and ARDS. Portable chest radiograph shows diffuse airspace disease with air bronchograms. Lung biopsy revealed pneumonia caused by *Pneumocystis carinii*.

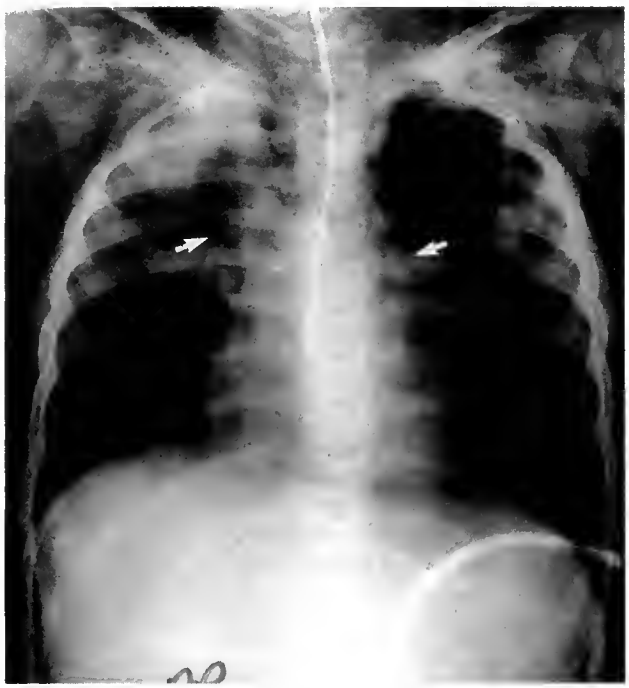

Fig. 40. Pneumomediastinum and subcutaneous emphysema. 6-year-old female with acute exacerbation of asthma. Anteroposterior portable chest radiograph shows extensive mediastinal and chest wall air as an "air block" complication of airway obstruction and alveolar rupture, with air dissection into the mediastinum. Note the thin mediastinal margins outlined on both sides by air (arrows). Air has dissected from the mediastinum up to the neck and over the shoulder regions. (*Figure continues on next page.*)

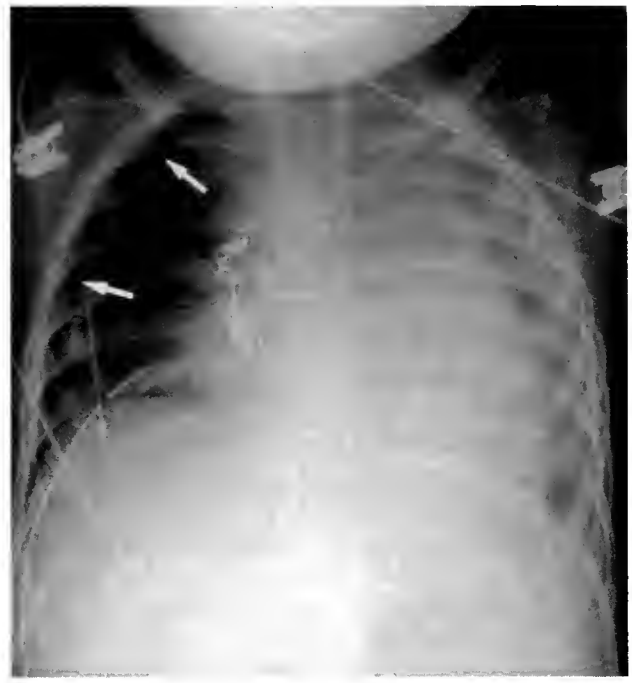

Fig. 41. Anteroposterior supine portable chest radiograph shows a moderate-size right pneumothorax (arrows), despite good positioning of a right pleural chest tube. In addition to cardiomegaly, there is partial collapse of both lungs, resulting in an increase in pulmonary opacification.

cysts or pneumatoceles, some of which may achieve large size.[91] Subpleural air cysts can cause pneumothorax if they rupture.[91] Etiologies of pneumatoceles include blunt chest trauma, staphylococcal pneumonia, and high-pressure mechanical ventilation with subsequent localization of air in the lung interstitium.[2,75,91]

Pneumomediastinum. Usually a reflection of barotrauma, air in the mediastinum of the PICU patient could also be caused by airway or intestinal (especially esophageal) rupture following chest trauma.[91] Pneumomediastinum, highly variable in appearance depending on quantity and location of mediastinal air, is detectable on the chest radiograph when air outlines the margins of anatomic structures not normally visible (Fig. 40).[75,91] Most pneumomediastinums are clinically benign and resolve spontaneously.[75,91]

Pneumopericardium. Pneumopericardium is usually a complication of mechanical ventilation in the premature newborn, blunt or penetrating chest trauma, or airway foreign bodies in the child.[75] Pneumopericardium is recognized radiographically as air surrounding the heart and is limited in its cephalad margin by the great vessels.[75] Because of the possibility of cardiac tamponade, pneumopericardium is potentially clinically important, and may require catheter or needle drainage.[16]

Subcutaneous Emphysema. Usually reflecting extension of a pneumomediastinum, subcutaneous emphysema presents on the chest radiograph as areas of streaky or confluent lucency around the neck and upper chest wall (see Fig. 40).[91] Although presenting both clinically and radiographically in a dramatic manner, subcutaneous emphysema is a benign process.[91] Air in the soft tissues of the neck in the absence of pneumomediastinum may signify injury to the upper airway during intubation or blind attempts at feeding tube placement.[91]

Pneumothorax. Pneumothorax due to barotrauma is a common emergency in patients on mechanical ventilation.[91] Pneumothorax may also result from blunt or penetrating chest trauma, invasive procedures such as central venous catheterization, endotracheal intubation, and feeding tube placement, or even in conjunction with pneumonia.[75,91] Both the quantity and distribution of pleural air are highly variable. Pneumothorax is most easily recognized on the AP supine chest radiograph as a zone of lucency separating the inner chest wall margin from the aerated lung (Fig. 41). If the patient is erect, the pleural air will collect at the lung apex. When the patient is supine, air will tend to collect in front of the lung and around the mediastinal

structures (an anteromedial pneumothorax).[91] The radiographic findings may include increased lucency of the involved hemithorax, a zone of relative lucency bordering the mediastinal structures and extending in a cephalo-caudal orientation, and unusually sharply-defined mediastinal borders.[75,91]

Air accumulating in the pleural space under increasing tension (greater than atmospheric pressure) is termed a tension pneumothorax.[75,91] Radiographic findings on the portable AP supine chest exam include a large pleural air collection, downward displacement with flattening or even inversion of the diaphragm, contralateral mediastinal shift, flattening of the heart border, and variable collapse of the ipsilateral lung (Fig. 42).[75,91]

Pleural Fluid Collections. Pleural fluid, whether reflecting an effusion, hemothorax, or empyema, appears as a homogeneous zone of soft tissue density separating aerated lung from the inner margin of the thorax (Fig. 43).[75] On an erect AP chest radiograph, pleural fluid will tend to localize at the lung bases, collecting lateral to or below the lung margins.[75,91] Frequent radiographic signs include blunting of the costophrenic angles, increased soft tissue beneath the lower lung margin, and soft tissue density in the lower lateral pleural space.[75,91] Pleural fluid collections are more difficult to identify on the AP supine chest radiograph because of the tendency of fluid to localize around the lung apex and the posterior basilar space.[91] In the former situation, a pleural soft tissue cap overlies the lung apex, while the latter distribution appears as a homogeneous increase in density of the lower thorax.[75,91] The decubitus AP chest projection is recommended to better evaluate questionable pleural fluid.[91] Chest CT will frequently identify subpulmonary effusions in the absence of radiographic signs of effusion on the AP supine chest radiograph.[91] Ultrasonography of the pleural space may confirm the presence of pleural fluid and further evaluate for associated changes such as adhesion, debris, and/or organization to suggest empyema or hemothorax rather than simple effusion.[91]

Chylothorax is an uncommon problem in childhood but can be seen with rupture of the thoracic duct secondary to blunt chest trauma and in the battered child syndrome.[91] Radiographic findings in chylothorax are similar to those of pleural effusion with regard to the pleural fluid collection itself.

Acute Chest Trauma

PICU patients with acute chest trauma may have experienced either penetrating injury (eg, gun shot or stab wound) or blunt trauma (eg, motor vehicle accident).[92]

Pulmonary Contusion and Hematoma. Resulting from either blunt or penetrating chest trauma, pulmonary contusion appears radiographically as either areas of homogeneous or nodular airspace consolidation or interstitial infiltration.[75,92,93] Usually present on the initial chest radiograph, pulmonary contusions may become larger and denser during the first 24–48 hours, then gradually resolve over a week or longer.[2,75,92] As they contract, pulmonary contusions may appear as a pulmonary mass.[75] Pulmonary hematomas usually appear round or oval from the onset.[2,75,93] Both contusions and hematomas may cavitate during resolution, resulting in formation of a pneumatocele or lung cyst.[2,75,93,94]

Pulmonary Laceration. Four types of pulmonary lacerations occur, based on mechanism of injury.[92] Type 1 lacerations, resulting from sudden compression of a pliable chest wall, reflect rupture of the air-containing lung, and appear radiographically as an air-filled linear structure or an intraparenchymal cavity, with possible associated

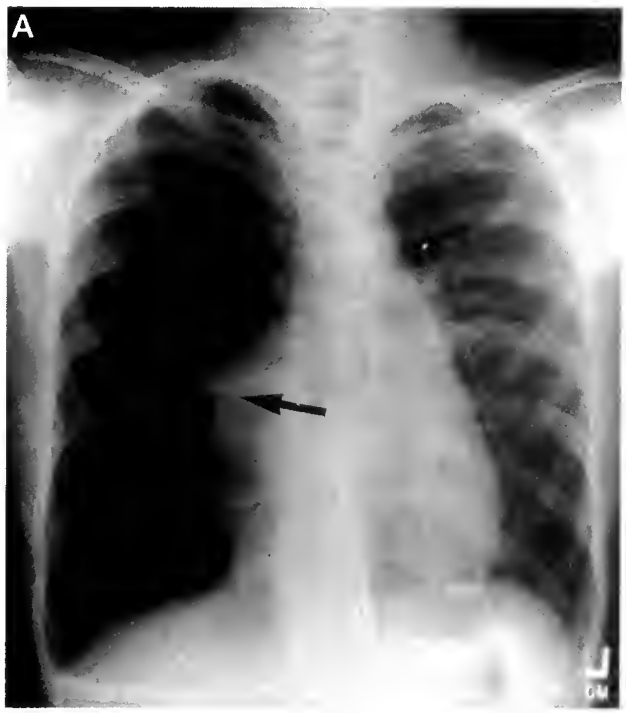

Fig. 42. Spontaneous pneumothorax with associated apical subpleural blebs. 14-year-old with sudden onset of shortness of breath and severe right-sided chest pain. A: Anteroposterior radiograph of the chest reveals complete collapse of the right lung (arrow) with a large pneumothorax occupying the entire right hemithorax and under at least mild tension, as indicated by depression of the right diaphragm and mild contralateral mediastinal shift. B: Computed tomography (CT) image after placement of a right pleural catheter (arrow) shows partial reexpansion of the right lung, but with considerable residual pneumothorax. C: CT image at lung apex shows several bilateral subpleural apical blebs (arrows), which predispose to pneumothorax. (*Figure continues on next page.*)

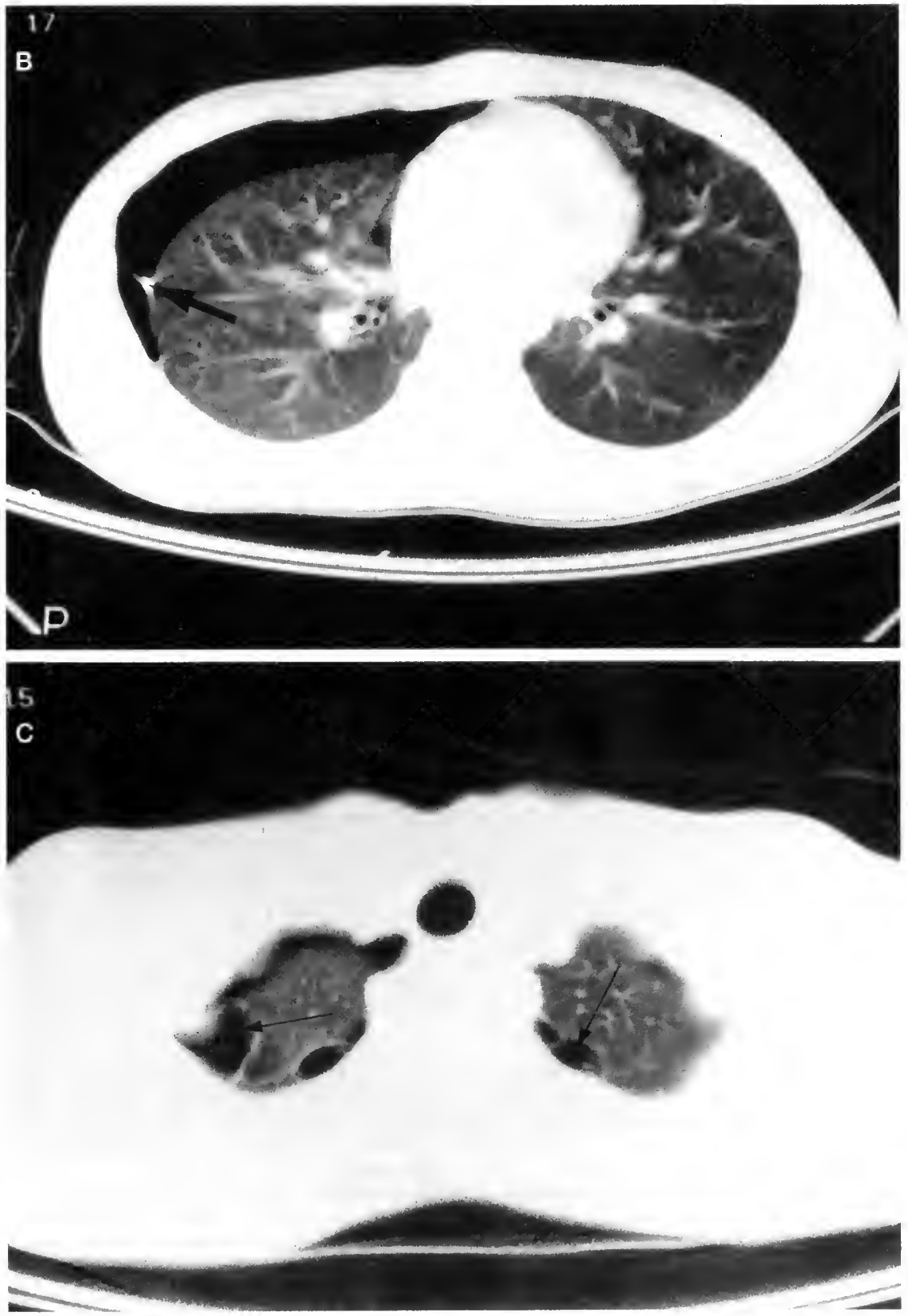

Fig. 42. *Continued*

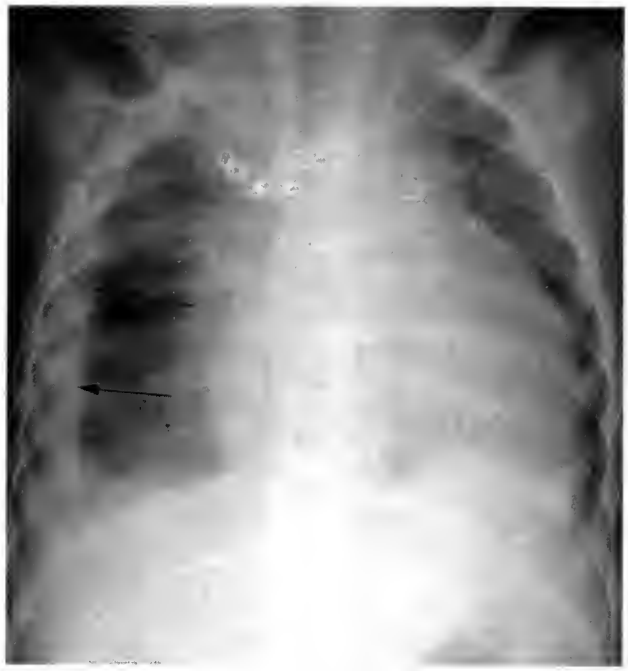

Fig. 43. Anteroposterior supine portable chest radiograph shows a large right pleural effusion (arrows) associated with decreased right lung volume. Also visible are sternal wires from prior midline thoracotomy, upper mediastinal surgical clips, and localized vascular coils in the right upper lobe for occlusion of collateral vessels.

pneumothorax.[92] Type 2 lacerations, which reflect a shear injury, appear as an air-filled pulmonary cavity in the paravertebral areas of the lower lobes.[92] Type 3 lacerations result from penetration of the lung by a fractured rib and appear as a small peripheral cavity or linear radiolucency with associated pneumothorax.[92] Type 4 lacerations occur in patients with pre-existing pleural adhesions, which cause the lung to tear when the chest wall is rapidly deformed or fractured; these lacerations are diagnosed only surgically or at autopsy.[92]

With bronchial fracture, massive pneumothorax and/or massive atelectasis may occur.[2,75,95] When a lung is completely detached from its bronchus, it may descend to the bottom of the hemithorax, which will be apparent on an upright chest radiograph.[75] With tracheal lacerations, pneumomediastinum or mediastinal hemorrhage is more common than pneumothorax, and an endotracheal tube may lie outside the trachea.[75]

Fractures of the Thoracic Skeleton. Severe chest trauma can result in fractures of the ribs, sternum, clavicles, scapulae, and thoracic spine.[92] Although common in conjunction with blunt chest trauma, rib fractures are often difficult or impossible to detect on the initial chest radiographs, unless displaced.[75] Radiographic evidence of a localized subpleural hematoma may indicate the presence of a nondisplaced rib fracture.[75] Rib fractures are more easily detected during the healing phase, when callus has formed at the fracture site.[75] Rib fractures may raise concern for more serious intrathoracic or abdominal injuries, such as great vessel disruption (associated with fractures of the first 3 ribs), splenic or liver trauma (of the anterior or lateral aspect of the lower ribs), or renal injury (of the lower posterior ribs).[2,75] Fractures of the sternum are best identified on the lateral chest radiograph and may be associated with underlying cardiac injury.[75]

Cardiovascular Injury and Mediastinal Hemorrhage. Severe chest trauma in children can infrequently result in transection or dissection injury to the thoracic aorta, resulting in mediastinal hemorrhage.[75,92] Chest radiographic signs of mediastinal bleeding in association with aortic injury include rightward displacement of the trachea and nasogastric tube (if present), obscuring of the margins of the aortic arch and descending aorta, a left apical soft tissue cap reflecting intrapleural fluid (hemorrhage), and widening of the mediastinum.[2,75,96]

Factors that complicate the identification of these findings include the presence of normal thymic tissue (in younger children especially) and the relative widening of the mediastinum that normally occurs on the supine chest radiograph.

The portable chest radiograph is limited in its ability to detect posttraumatic cardiac injuries such as bloody or serous pericardial effusions (Fig. 44), myocardial contusions, or traumatic cardiac aneurysm.[75] Enlargement of the cardiac silhouette may be the sole chest radiographic finding with these complications.[75]

Pulmonary Embolism

Pulmonary embolism due to vascular thrombosis is an uncommon problem in children, but is generally underdiagnosed.[97] Risk factors for pulmonary embolism include shunted hydrocephalus, trauma, heart disease, infection, and neoplasia.[97] Although in pulmonary embolism the chest radiograph is frequently normal, it may show focal areas of diminished pulmonary blood flow (oligemia), pleural effusions, and, rarely, a focal pleural-based, wedge-shaped consolidation ("Hampton's hump") that reflects the resultant pulmonary infarct.[98]

Fractures of marrow-containing bone can result in the intravascular dispersal of fat emboli, with uncommon but potentially serious pulmonary, cerebral, and cutaneous manifestations in children.[75,92] The chest radiograph, initially normal, can show patchy opacities and then widespread diffuse opacity resembling pulmonary edema within about 72 hours of injury. Air bronchograms are often present, while heart size is typically normal.[92]

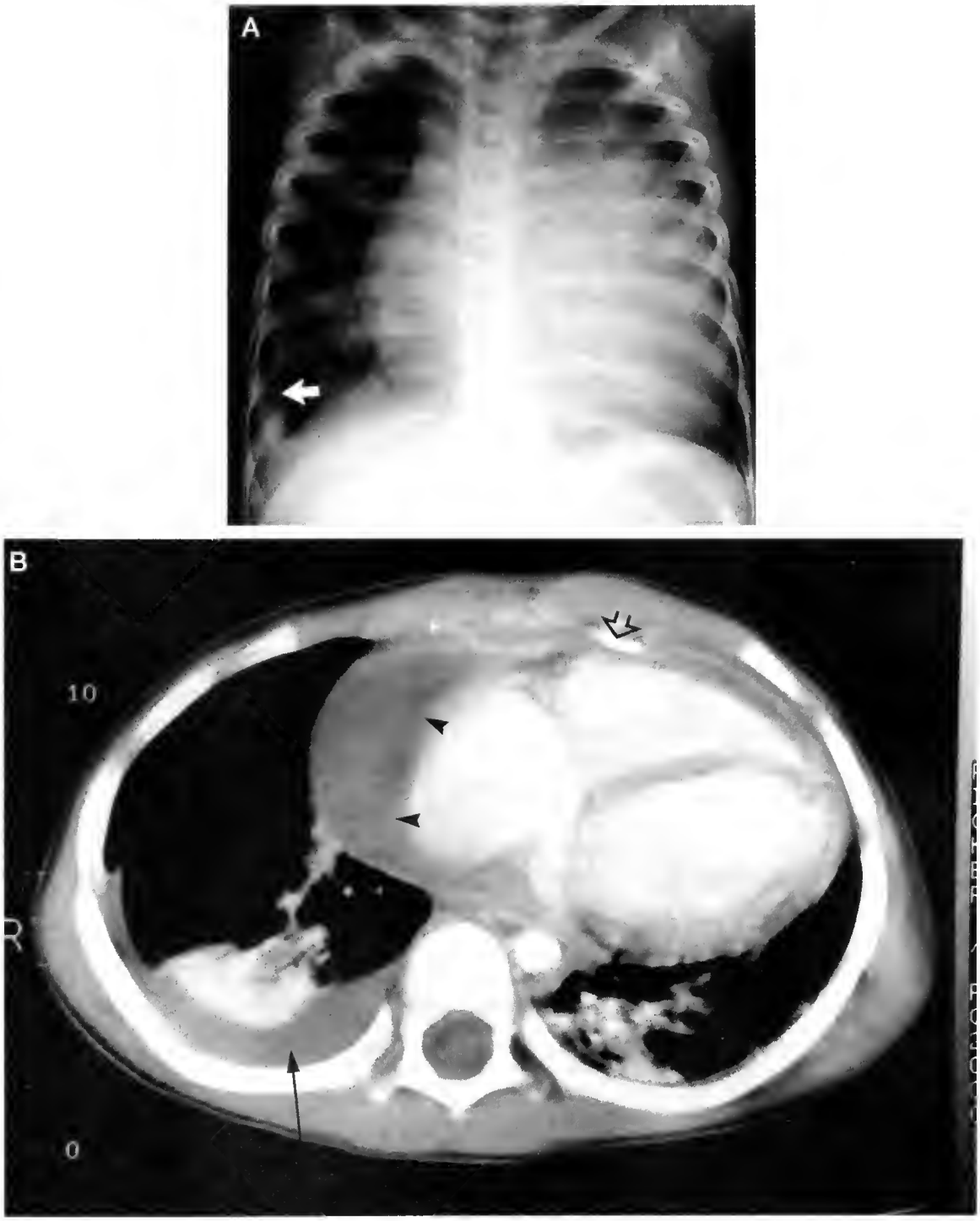

Fig. 44. Three-year-old male with thoracic trauma. A: Anteroposterior supine chest radiograph shows marked enlargement of the cardiac silhouette, compatible with cardiomegaly and/or pericardial fluid. The chest radiograph cannot reliably differentiate between cardiac dilatation and fluid in the pericardial space. A right pleural effusion (arrow) is present. B: Contrast-enhanced computed tomography image through the lower chest shows pericardial fluid, predominantly on the right (arrowheads), atelectasis of both lower lobes, with an associated right pleural effusion (arrow) and a catheter entering the pericardial space anteriorly (open arrow). Contrast permits differentiation of the cardiac chambers from the pericardial fluid.

Summary

Both neonatal and pediatric patients with an acuity of illness requiring monitoring and support in an ICU will typically need some line, tube, and catheter connection, any of which are subject to possible malposition or other complications. The portable chest radiograph provides invaluable assistance in determining whether these devices are properly placed and whether certain complications are present or possible.

In addition, the numerous disease processes involving or affecting the thorax and contents can be monitored and evaluated fully only with the contribution of the chest radiograph. Attention to proper radiographic technique increases the yield of meaningful diagnostic information provided by the portable chest radiograph in these patients.

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Uses of Thoracic Computed Tomography in the Intensive Care Unit

Wallace T Miller Jr MD

Introduction
Chest Radiography versus Computed Tomography
Mediastinum
Chest Wall
Lung Parenchyma
Pleura
Trauma
Abdomen
Computed Tomography's Influence on Diagnosis and Care Plan
Summary

[Respir Care 1999;44(9):1127–1136] *Key words: computed tomography, radiography, intensive care unit, mediastinum, complications, diagnostic accuracy.*

Introduction

Patients in the intensive care unit (ICU) are critically ill, with a high mortality rate. Care of these patients is a complex task and in many cases there are nonspecific clinical findings such as fever or respiratory failure without clear cause. In this setting, imaging can play an important role in obtaining a specific diagnosis or in directing further evaluation in order to obtain a diagnosis. Chest radiography plays an important role in the evaluation of the thorax but has known limitations, especially in this critically ill population.[1–3] Thoracic computed tomography (CT) is more sensitive than chest radiography in detecting subtle lung parenchymal abnormalities. CT is also known to improve evaluation of mediastinal, pleural, and chest wall processes, compared to chest radiographs.[4] However, despite these theoretical advantages, there have been few published studies directed toward ascertaining the utility of thoracic CT in the ICU.[4–8] These few studies include retrospective reviews of 87 CT exams of patients in a trauma ICU,[5] 20 patients in a general ICU,[5] and 108 CT exams of patients in 3 adult ICUs, the surgical, medical, and cardiac ICUs.[8]

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Chest Radiography versus Computed Tomography

My colleagues and I compared CT findings to chest radiographic findings and found that, if disagreements about mild congestive heart failure are excluded, they agree in 93% of findings.[8] This high accuracy of the chest radiograph for basic findings in the thorax confirms the utility of chest radiography in this clinical setting. However, though we found chest radiography to be relatively accurate regarding the findings it detects, it was insensitive to many of the findings detectable by CT. The chest radiograph detected only 48% of the findings discovered by thoracic CT.

In our study, CT of the thorax showed new information in the vast majority of exams in intensive care patients. However, the majority of these findings had little "clinical significance." Small pleural effusions, small pneumothoraces, and minor lower lobe atelectasis accounted for 52% of these new findings.

Thus, although the CT can often identify new findings, this is not necessarily sufficient justification for performing thoracic CT in the ICU. Therefore, it is necessary to look at the frequency and type of clinically significant findings in order to begin to evaluate the utility of thoracic CT in the ICU. All 3 major studies of CT in the ICU found CT to provide important information for critically ill ICU patients. Mirvis et al found that of 87 thoracic CT scans, 70% yielded "significantly more diagnostic information" than chest radiographs.[4] Golding et al felt that 75% of thoracic CT scans in 20 ICU patients were "judged useful by subsequent clinical course when they indicated main-

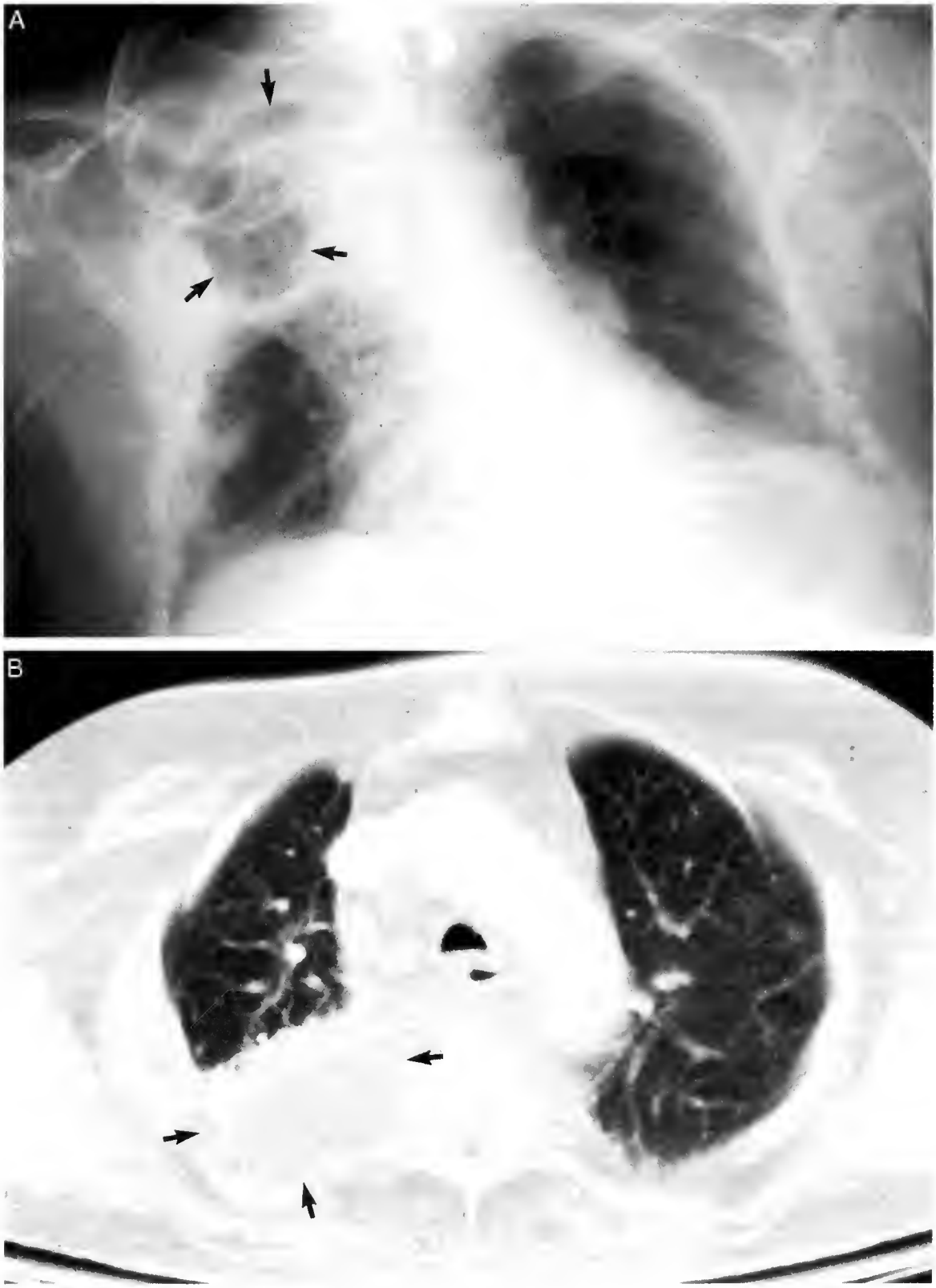

Fig. 1. A 68-year-old woman with persistent fever. A: Chest radiograph shows an apparent cavitary infiltrate in the right upper lobe, thought to represent a lung abscess. B: Computed tomography scan shows the abnormality to be a loculated pleural effusion and associated passive atelectasis, without evidence of cavitation.

Fig. 2. An elderly man admitted to the cardiac care unit for angina. A: Admission chest radiograph suggests multiple pulmonary nodules. B: Computed tomography scan confirmed multiple pulmonary nodules (arrows), subsequently found to be metastases.

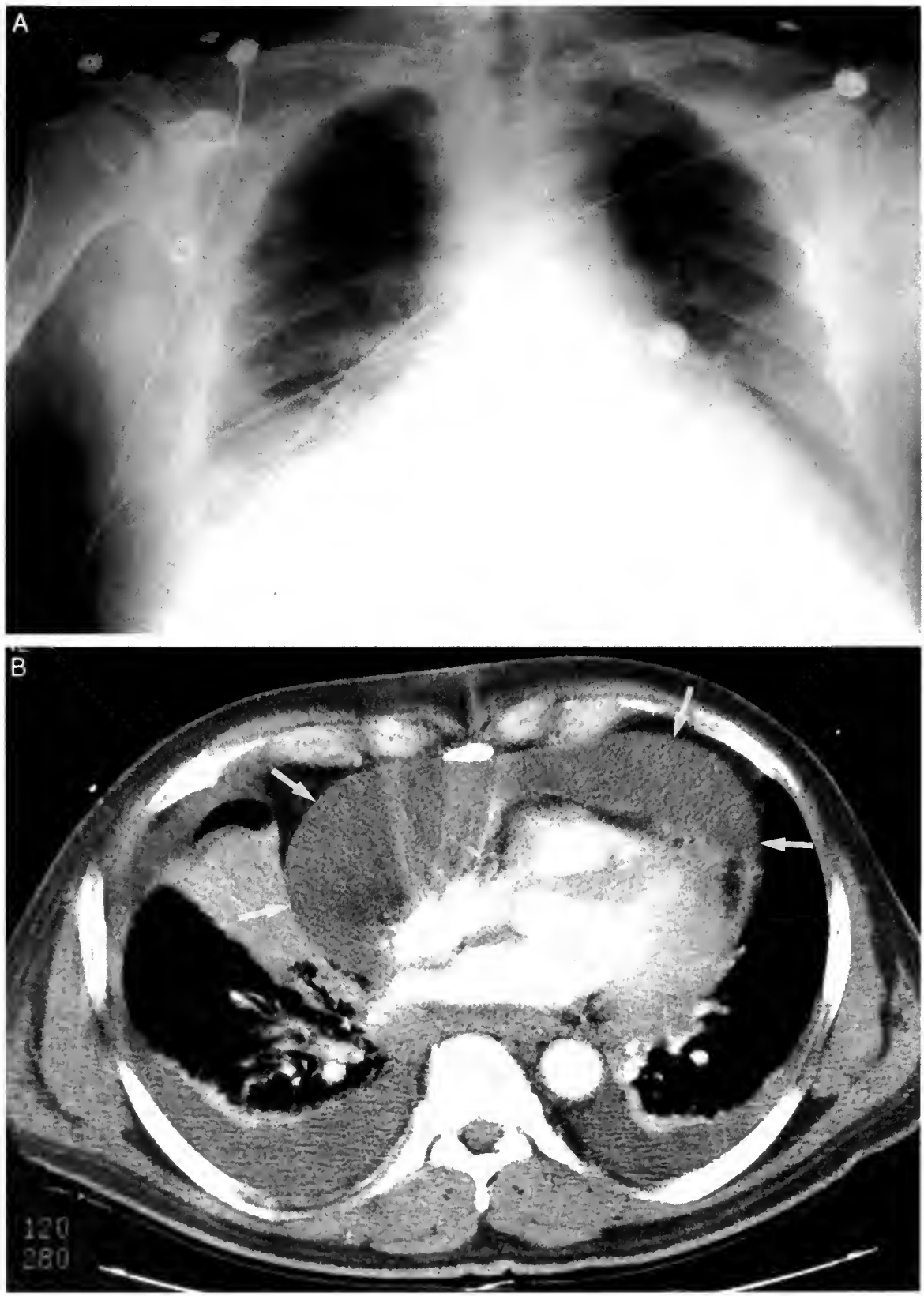

Fig. 3. A 60-year-old man with a history of lung carcinoma was admitted to the cardiac care unit with severe dyspnea and leg edema. A: Chest radiograph shows enlarged cardiac silhouette and small bilateral pleural effusions. B: Computed tomography scan shows an unsuspected pericardial effusion compressing the right ventricle. The diagnosis was pericardial tamponade, and the patient underwent emergency pericardial drainage.

Fig. 4. This man remained persistently febrile following esophagectomy and gastric pull-through. A: Chest radiographs were interpreted as showing bilateral aspiration pneumonia. Despite adequate broad spectrum therapy for aspiration pneumonia, fever did not improve. B: Computed tomography (CT) scan shows a gastric-pleural fistula (curved arrows) and empyema (straight arrows) that were not detected on the prior chest radiographs. C: CT scan also allowed identification of gas dissecting through the paraspinal musculature (arrow), indicating a necrotizing fasciitis. Review of the CT scan led to emergency fasciotomies and pleural decortication.

tenance or alteration of existing treatment strategies."[5] Miller et al found that "72% of exams demonstrated clinically significant information."[8]

Clinically significant information is of 3 types: (1) negation of particular clinical concerns (eg, rule out aortic dissection or rule out postoperative fluid collection) (Fig. 1), (2) affirmation of diagnoses suspected by prior clinical or radiographic findings (Fig. 2), or (3) demonstration of new, unsuspected findings.[8] A negative conclusion by CT was the most often clinically significant finding in 45% of CT scans. In the absence of new findings, this might indicate maintenance of the current clinical management. Eleven percent of thoracic CT exams confirmed an abnormality suspected by prior chest radiographs. Thirty-one

Fig. 5. Three weeks after coronary artery bypass grafts, this patient was persistently febrile. Chest radiographs (not shown) indicated usual postoperative changes and provided no explanation for the fever. Chest computed tomography shows diastasis of the sternal incision, with an intervening fluid collection, findings associated with sternal osteomyelitis, and an associated chest wall-mediastinal abscess.

percent of CT exams demonstrated a clinically significant abnormality that was not expected on the basis of the chest radiograph.

Mediastinum

The mediastinum is the most common site of clinically significant abnormalities in general ICUs (33–34%).[5,8] In a trauma ICU, mediastinal abnormalities are less common, and were seen in only 1 of 87 exams in a study by Mirvis.[4] Unexpected fluid collections or postoperative complications were the most common significant abnormality in the mediastinum (Figs. 3, 4 and 5). Detection or staging of mediastinal involvement by malignancy was another important use of CT in this population (Fig. 6).

Chest Wall

Like the mediastinum, the chest wall is relatively poorly-evaluated by chest radiography. However, significant chest wall abnormalities are rare in ICU patients, found in 0–5% of exams.[4,5,8] Clinically significant chest wall findings usually are related to abnormal fluid collections or other chest wall infections, or to the spread of malignancy (see Figs. 4 and 5).

Lung Parenchyma

Pneumonia and lung abscess are 2 other unsuspected and clinically significant abnormalities found in 6–15% of exams (Fig. 7).[4,5,8] Evaluation of the pulmonary parenchyma in ICU patients is unlike the same evaluation in the outpatient population. The intensive care patient is likely to have combinations of multiple thoracic problems, such as pulmonary edema, atelectasis, pneumonia, and pleural effusions. These can result in superimposed thoracic opacities and one or more processes can be obscured. The CT can provide greater tissue differentiation, which may alter the interpretation of pulmonary opacity. CT interpretation also benefits from the absence of superimposition of thoracic structures.

Pleura

CT was instrumental in management of pleural abnormalities and accounted for 17–30% of clinically signifi-

Fig. 6. A 60-year-old man with a history of coronary artery disease and emphysema complained of near-incapacitating dyspnea. Computed tomography (CT) scan shows a large intratracheal mass that nearly occludes the lumen of the trachea. This mass was not recognized on the prior chest radiograph (not shown), and was found to be an adenoid cystic carcinoma. CT was helpful in delimiting the extent of the tumor for surgical excision.

cant new findings.[4,5,8] These include unsuspected pleural effusions, empyemas, and pneumothoraces (Figs. 1, 4, and 8). Pneumothoraces were an important finding predominantly in the trauma population. Ultrasound examination would have probably provided (at a lower cost) information equal to CT with regard to some pleural abnormalities. However, ultrasound is a unidimensional tool with regard to the thorax. Clinicians often prefer the more complete thoracic examination afforded by CT.

Trauma

Mirvis et al's study of a trauma ICU noted that detection of malpositioned or clogged chest tubes is an important and frequent finding of thoracic CT; 15% of their exams noted some abnormality of thoracostomy tubes.[4] In the studies by Golding et al, and in my own study, this was an uncommon finding, in 5% and 2% of exams, respectively.[5,8] This difference in studies likely reflects differences in the patient populations. In the trauma setting, thoracostomy tubes must be inserted in a rapid percutaneous fashion that is more prone to misplacement than in the setting of surgery or elective drainage of pleural effusions, as is more prevalent in a general ICU.

Abdomen

Thoracic CT often has the unintended result of evaluating the upper abdomen. In the study performed by me and my colleagues, 6% of exams found clinically relevant abnormalities below the diaphragm (Fig. 9).[8]

Computed Tomography's Influence on Diagnosis and Care Plan

One of the most stringent criteria for the utility of any test is the frequency with which it changes clinical management. Studies have shown that 22–25% of thoracic CT scans have resulted in changes in management.[8–10] In one study, over half of these interventions involved drainage of pleural, pericardial, chest wall, or retroperitoneal collections.[8]

The ultimate measure of any clinical intervention is its effect on the morbidity and mortality of patients. To date, no studies have been able to measure thoracic CT's effect on ICU patient morbidity and outcome. Furthermore, while CT is a powerful tool in the evaluation of the thorax, and research has shown a variety of ways that CT can be clinically useful, it is important to remember that, like all tests, there will be an incidence of false positive and false negative exams. The magnitude of these problems has also not been measured.

It is also important to note that the study populations to date have been highly selected groups, so the above results do not necessarily reflect the utility of thoracic CT for ICU patients as a whole. What results indicate is that thoracic CT is useful for the clinical indications for which it was performed. Those indications are primarily (1) the evaluation of fever or sepsis of unknown cause, (2) search for postoperative complications, (3) characterization of pleural effusions, and (4) detection and staging of thoracic malignancies.[8]

Summary

CT can play an important adjunctive role to portable radiography in the thoracic evaluation of certain ICU patients. In the majority of patients, findings not detected by portable chest radiography are discovered by CT. More than half of thoracic CT exams provide additional information important in understanding a patient's clinical situation, and result in changes in clinical management in approximately one fifth of patients.

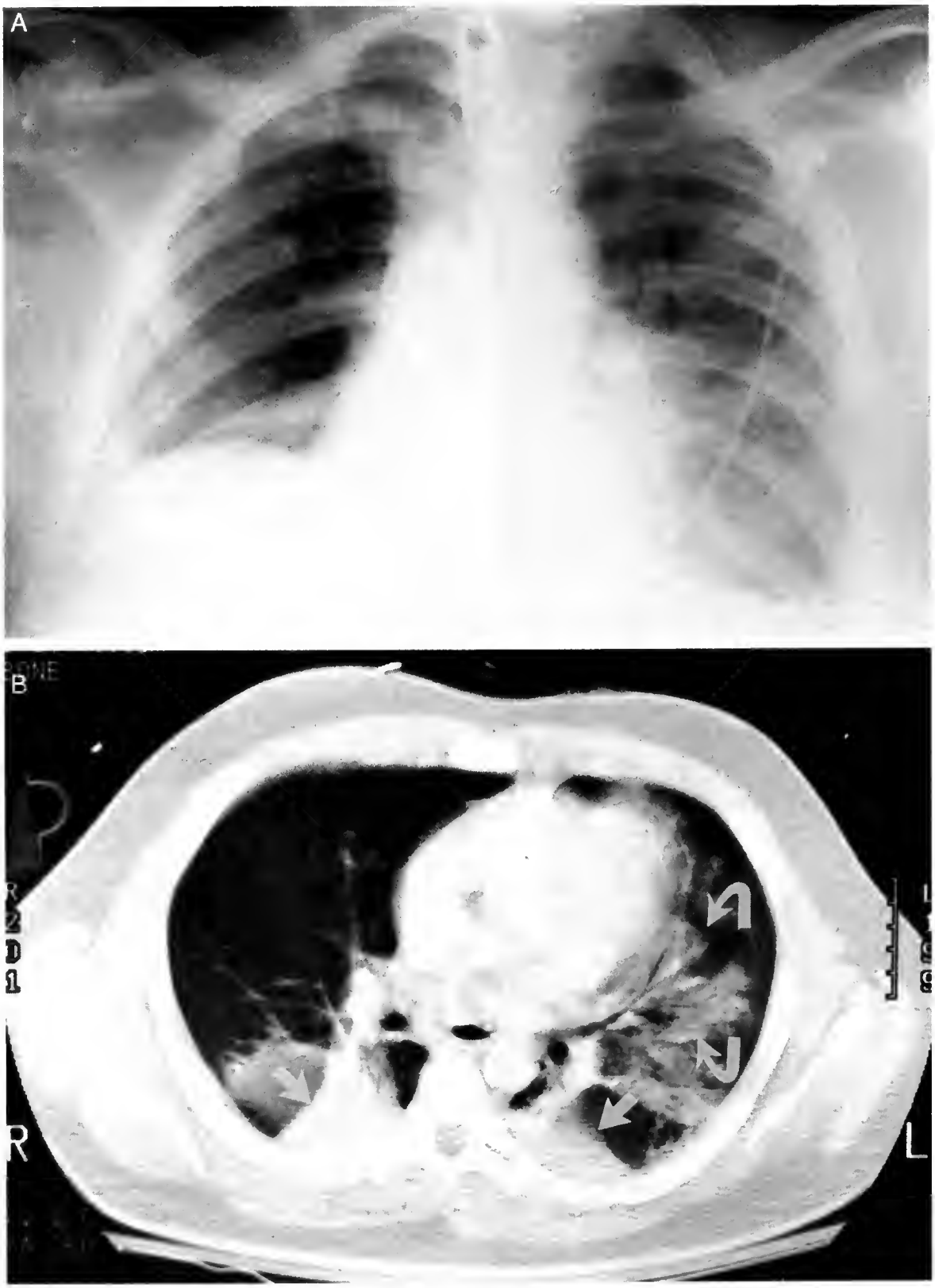

Fig. 7. Young man who, following an automobile accident, remained comatose and persistently febrile without clear cause. A: Numerous chest radiographs before and after the onset of fever were unchanged from this radiograph. All were interpreted as showing stable bibasilar atelectasis. B: Computed tomography confirms the presence of atelectasis as 2 dense triangles of pulmonary opacity (straight arrows) in the lower lobes, and also reveals unsuspected pneumonia in the lingula (curved arrows).

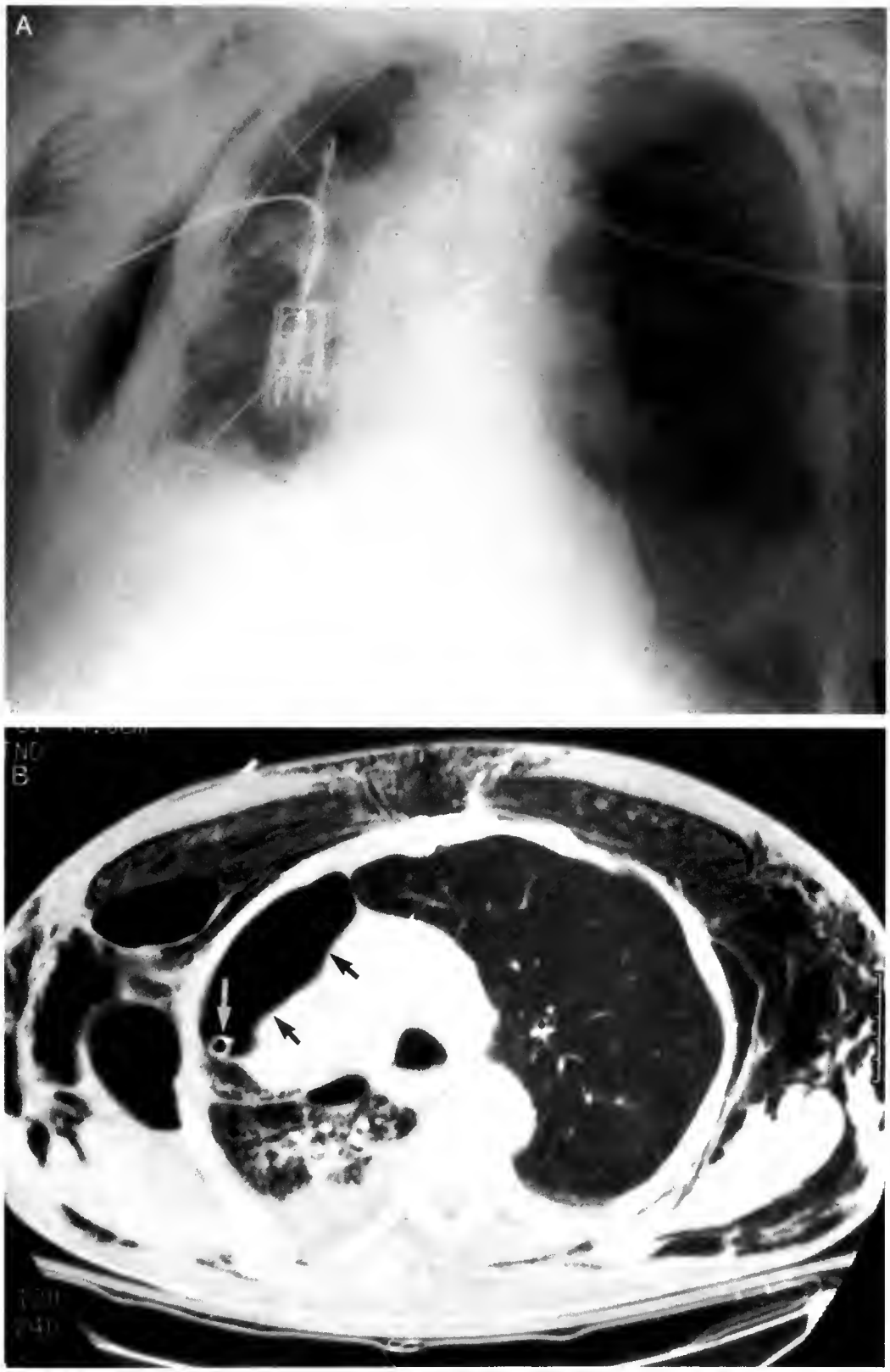

Fig. 8. One week after right upper lobectomy. A: This chest radiograph and multiple prior studies were interpreted as showing patchy right lung atelectasis and subcutaneous emphysema, but no evidence of pneumothorax. Despite absence of a radiographically-identifiable pneumothorax, this patient had a persistent high volume air leak in his thoracostomy tubes. B: Computed tomography shows a > 50% pneumothorax (black arrows). The pneumothorax resolved with the addition of another thoracostomy tube and manipulation of the existing tubes (white arrows).

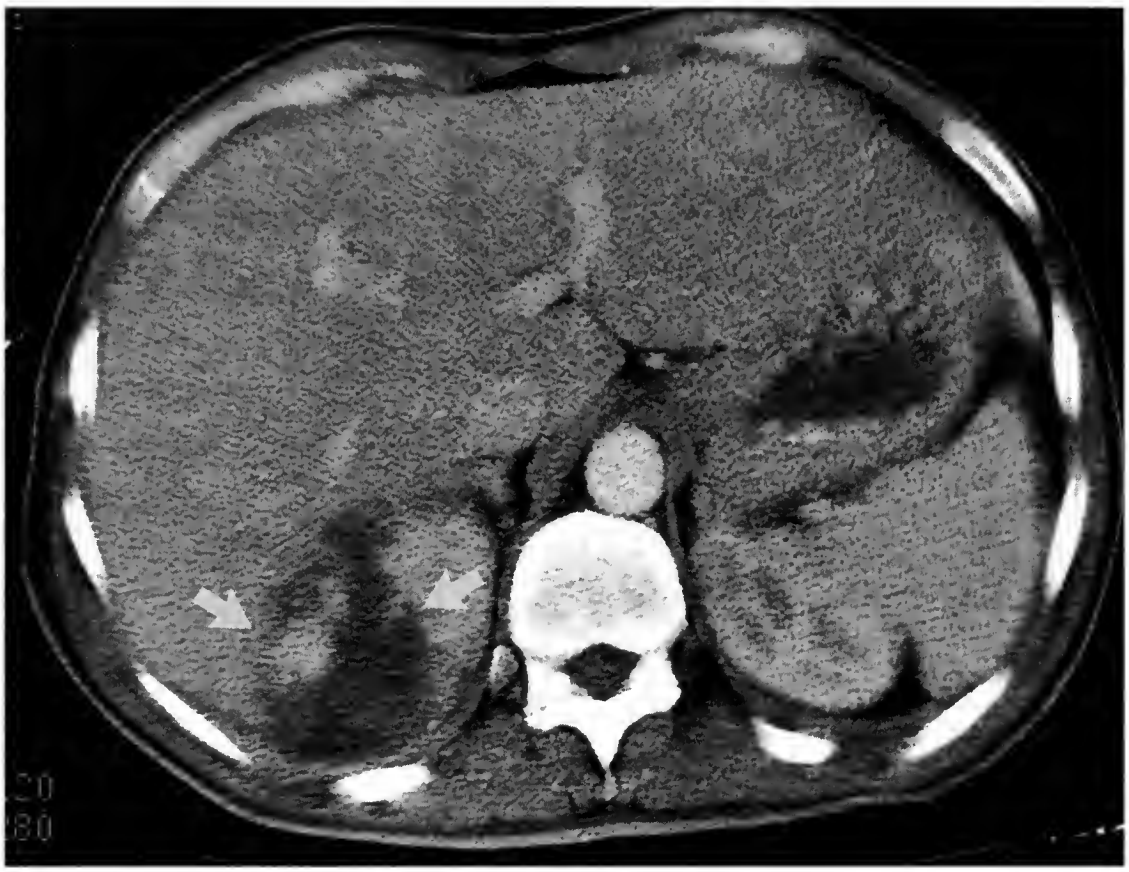

Fig. 9. A 43-year-old man who was an intravenous drug user had persistent fevers. Chest radiograph (not shown) was interpreted as normal. This computed tomography scan of the chest showed a cavitary mass in the upper pole of the right kidney, which was found to represent a renal abscess secondary to staphylococcal endocarditis.

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Books, Films, Tapes, & Software

Thoracic Radiology: The Requisites, Theresa C McLoud MD, Editor. Hardcover, illustrated, 541 pages. St Louis: Mosby; 1998. $79.00.

Interpreting a chest radiograph is one of the most common and potentially challenging tasks facing the clinician. Subtle differences in the texture of a lung opacity or the shape of a mediastinal contour can suggest remarkably different diagnoses and therapeutic plans. Learning and retaining the knowledge needed to correctly interpret a chest imaging study can be a daunting, ongoing task for physicians, radiologists, and nonradiologists alike. **Thoracic Radiology: The Requisites** presents a comprehensive, well-organized, in-depth approach to analyzing chest imaging studies and formulating an appropriate differential diagnosis based on the radiographic findings.

This book, edited and partly written by Dr McLoud, has a total of 5 contributors, all leading thoracic radiologists. There are 19 chapters. The first chapter describes a general approach to chest imaging, including a review of thoracic anatomy, that every physician will find very helpful. The next 5 chapters address special topics such as congenital anomalies, infections in normal and immunocompromised hosts, intensive care unit patients, and trauma. The next 8 chapters discuss specific pulmonary and bronchovascular disease processes. The remaining chapters discuss the mediastinum, pleural disease, and interventional radiology techniques for the thorax.

The text is clear, complete, and very readable. There are numerous well-organized tables of differential diagnoses for common radiographic findings. The radiographic reproductions are very good and illustrate classic examples of each finding. Where appropriate, plain film findings and disease processes are correlated with either conventional or high-resolution computed tomography images.

This book is well suited to its goals of providing core educational material for radiology residents, as well as serving as a reference and refresher for practicing radiologists. Medical students and nonradiology residents will probably find the detail and scope of this book excessive, but any clinician looking for either an in-depth understanding of thoracic imaging or a good reference volume will find this book very helpful.

The book is well referenced, and contains very good quality images. As with all of the books in the "Requisites" series, production quality is excellent. I highly recommend this book for any medical school or departmental library. It will also be a welcome addition to the library of any clinician who regularly interprets chest radiographs.

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Principles of Critical Care, 2nd edition, Jesse B Hall MD, Gregory A Schmidt MD, and Lawrence DH Wood MD PhD, Editors. Hardcover, illustrated, 1,767 pages. New York: McGraw-Hill; 1998. $175.00.

This comprehensive and highly organized textbook of critical care medicine provides an in-depth discussion of diseases and complex issues related to the intensive care unit (ICU). The book is organized into 11 parts and each part is divided into chapters. Part I provides an overview of the approach to and organization of critical care in a modern ICU and includes chapters discussing the team approach, economics of critical care, infection control, assessment of severity of illness, and transportation of the critically ill patient. Part II discusses the general management of the ICU patient, including chapters on sedation and paralysis, airway management, hemodynamic monitoring, nutrition, withdrawal of life support, and an up-to-date chapter on ventilator-induced lung injury. Parts III through IX include organ-specific discussion of diseases, covering the wide spectrum of illnesses seen in a cardiac ICU, medical ICU, neurology ICU, and bone marrow transplant unit. Part X discusses the surgical patient, including the preoperative assessment of the high-risk patient, transplantation, trauma, and burns. This part also includes a very useful chapter on interventional radiology in the ICU. Part XI focuses on all the special problems that deserve separate discussion and consideration, such as toxicology, critical illness in pregnancy, critical care pharmacology, diving medicine, near-drowning, and others.

This book would be most useful for physicians, especially those intending to pursue a career in critical care medicine, yet certain sections of the book can prove valuable for respiratory therapists and nurses working in an ICU or intermediate care setting. Parts I and II especially could be useful for therapists and nurses, particularly the chapters discussing the team approach, economics of critical care, infection control, sedation, paralytics, airway management, respiratory system monitoring, early detection of complications of critical care, and withdrawal of life-sustaining therapy. A chapter on transportation of the critically ill patient discusses new treatment modalities that have significant impact on the feasibility and tactics of transport, such as noninvasive ventilation and heliox, which I think will be very useful for all ICU personnel. Other chapters useful for therapists and possibly nurses include those on ventilator-induced lung injury, noninvasive ventilation, and liberation from mechanical ventilation.

The authors/editors have succeeded in their aims of providing a comprehensive and up-to-date discussion of critical care medicine in a well-organized and easy-to-read book. The gradual transition of the parts from general issues to specific issues, and the clear writing and organization within the chapters will be particularly valuable to physicians in training, early in their exposure to critical care. Every chapter begins with key points, summarizing the chapter's themes and take-home messages, and ends with an illustrative case and discussion.

The solid grounding in pathophysiology present in the first edition is preserved in this edition, and is evident at the beginning of most chapters and especially in 2 dedicated chapters. The chapter on pathophysiology of the circulation in critical illness provides an excellent discussion of pump function and factors controlling venous return. It also discusses the pulmonary circulation and factors governing lung liquid flux. Interpretation of pulmonary artery catheter measurements and interactions between respiration and circulation are highlighted as a basis for understanding the discussion of the pathophysiology of

cardiovascular and pulmonary disorders in the subsequent chapters. The chapter on pathophysiology and differential diagnosis of acute respiratory failure also illustrates solid grounding in pathophysiology. The different types of respiratory failure and the basic pathophysiologic principles leading to each type are explained in preparation for the more specific discussions that follow.

The arguments presented are clear and the authors/editors demonstrate appropriate skepticism based on scholarly review of the literature before arriving at their conclusions. The chapter on acute hypoxemic respiratory failure is an example of this approach. In the discussion on salvage therapies for acute respiratory distress syndrome—such as prone positioning, high-frequency ventilation, and extracorporeal membrane oxygenation—the authors objectively describe the reasoning behind each therapy and the data for beneficial and nonbeneficial outcomes. They then report possible special circumstances where the therapy may be useful. The same applies to the part on nitric oxide and corticosteroids in acute respiratory distress syndrome.

An outstanding addition to this book is the inclusion of exciting chapters dedicated to hot new topics such as ventilator-induced lung injury, noninvasive ventilation, acute right heart syndromes, systemic viral infections (eg, the genus hantavirus and *Ebola* virus), and multisystem organ failure. The chapter on acute right heart syndromes is extremely interesting and would have been perfect if the authors had mentioned the controversy regarding the pathophysiology of acute right ventricular failure. The authors describe data supporting demand-supply mismatch and subsequent ischemia as the major insult to the right ventricle. Though this is part of the insult, some investigators believe other factors play a role in a complex interrelationship. The chapter on multisystem organ failure contains a wealth of information, including many figures, tables, and drawings explaining this complex syndrome and its sequence of events, both from organ to organ and at the cellular and molecular level.

The book is elegant, the choice of titles self-explanatory, and the script clear. The quality of the illustrations is good and for its price of $175 it is a very good bargain, considering the wealth of information it provides. I would highly recommend this book as an addition to ICU libraries. It would be particularly helpful for residents and fellows planning a career in critical care, and parts of this book will also prove very useful for therapists and nurses in the critical care setting. It is a most welcome addition to my library.

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The ICU Book, Paul L Marino MD PhD. Soft-cover, illustrated, 928 pages. Baltimore: Williams & Wilkins; 1998. $55.00.

Intensive care medicine has been one of the fastest growing of all medical specialties. Intensive care units (ICUs) in hospitals today house the sickest patients from all departments. Medical staffing of ICUs varies from internists and surgeons trained specially in critical care medicine, to anesthesiologists trained likewise, to "pure intensivists" who practice solely in ICUs. The patient care teams are truly multidisciplinary, with nursing, respiratory therapy, nutritional support, and physical therapy all making important contributions. The disease spectrum varies widely and is ever growing. With technologic advances affecting all fields of patient care, newer pathological and physiologic states are encountered that are unlike any previously described. In the maze of this growing field, with controversies and uncertainty surrounding every aspect of care, well-reviewed literature is always appreciated. Several well-referenced textbooks exist in the field, but unfortunately they tend either to be too lengthy or to address only one particular aspect of intensive care. Here the author takes a unique approach by writing an entire book himself and trying to cover all aspects of intensive care. To a great extent, he succeeds in his efforts.

The ICU Book tries to address all aspects of patient care in the ICU. The author states at the outset that the book does not cover burns, trauma, or obstetrics, as these are "highly specialized" areas of intensive care. The author takes a physiology-based approach to care, trying to define the pathophysiology of disease processes and then explaining the therapy based on that. The book is divided into 15 sections. The author takes a nontraditional approach to these sections but follows the general scheme of describing the physiologic basis early on, then procedures, followed by the systems approach so prevalent in ICUs. Nearly half of the book discusses cardiopulmonary issues. Then comes a discussion of general principles of management of infectious diseases. The next section is on acid-base disorders, where the reader will find care of diabetic ketoacidosis. This is followed by approaches to electrolyte and hematologic disorders. After discussing other systems and pharmacology, the book ends with interesting appendixes. Each chapter includes references that have been nicely categorized. The reader will be quickly able to find out some of the reviews on the subject, separate from original studies. Most chapters are to the point and have appropriate tables and figures as and when necessary. An index is provided and is arranged well. The book is balanced; this is a commendable achievement by a single author.

I found 2 or 3 grammatical errors. Of note were 2 factual errors that remind you of the importance of being sure of measurement units and decimal points in numbers while working in the intensive care environment. On page 305, it should read that thrombolytics are indicated for ST segment elevation of more than 1 mm (not 0.1mm) in 2 contiguous leads. The unit for V_{CO_2} on page 351 should be mL/min/M^2, and not L/min/M^2.

I do have some points that I would like to ponder. Although the author has largely avoided bias of individual opinion, it shows up from time to time. If you use the book as the sole review of the subject, you will be getting one individual's opinion in certain aspects, like the author's bias against corticosteroid use in chronic obstructive pulmonary disease exacerbation, or the author's preference for using bowel decontamination solution. Not that these are controversial issues, but that the references quoted on these and some other topics substantiate only the author's practice style and do not represent both sides of controversies. Some of the discussions (eg, how to decide the amount of magnesium replacement) appear too lengthy. I enjoyed the appendixes but did not understand the need for the world population growth rate table and other general tables in this book. I would add to the list of "highly specialized" areas of intensive care such complex situations as post-transplantation (solid organs) or some of the postoperative care that takes place in ICUs these days, such as double lung ventilation and left ventricular assist devices.

I feel the book would be very helpful for beginners, such as medical students or interns, to begin to appreciate the complexity of critically ill patients. Respiratory therapists will find the discussions on oxygen transport and delivery helpful. Mechanical ventilation is covered only modestly. For nurses and other practitioners this book provides a wealth of information about critical care in easily readable format. For physicians and nurses trained in intensive care, the book would be an easily accessible review material. I said review. You should have a view of the subject to review it. **The ICU Book** would be a good addition to your "portable brain" but is not a replacement of your standard textbook on the subject.

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Basic ECG Interpretation, URL: www.saminc.com/ecg.course.htm. Internet-offered 25-hour credit course, offered by Fanshawe College, London, Ontario, Canada, and written and published by Internet-Based Learning, London, Ontario, Canada; 1998. Registration Fee $103.00 Canadian, $65.00 U.S.

This Internet course is composed of 10 units of instruction: (1) anatomy and physiology, (2) electrophysiology, (3) electrocardiogram (ECG) recording paper and leads, (4) sinus rhythms, (5) atrial rhythms, (6) junctional rhythms, (7) ventricular rhythms, (8) atrioventricular blocks, (9) pacemaker rhythms, (10) clinical rounds. There is a formative section examination at the end of each unit, as well as a final comprehensive summary exam that can only be accessed after successful completion of the sectional examinations.

The first 2 units thoroughly review the anatomy, physiology, and electrophysiology necessary to fully understand the applications that comprise the next 8 units. These introductory units cover such topics as structures of the mediastinum, cardiac chamber anatomy, neurotransmitters, muscle characteristics, the cardiac conduction system, action potentials, the sodium pump, and basic causes of arrhythmias.

Unit 3 concentrates on the practical side of ECGs. It details paper calibration and limb placement, and provides visual representations of the cardiac cycle. Unit 4 covers the sinus rhythms, including bradycardia, tachycardia, blocks, and arrests. The atrial rhythms (premature atrial complexes, supraventricular tachycardia, atrial flutter and atrial fibrillation) are discussed in Unit 5. Unit 6 reviews a wide range of junctional rhythms. Unit 7 describes ventricular rhythms. Of particular note are premature ventricular complexes, escape beats, ventricular tachycardia, ventricular fibrillation, and asystole. This section also identifies various medications used in the treatment of dysrhythmias.

Units 8 and 9 present a comprehensive analysis of the major atrioventricular blocks and pacemaker rhythms. The final instructional section, Unit 10, provides a clinical review of ECG analysis, plus several case studies that highlight the major cardiac arrhythmias covered in the previous sections.

This instructional tutorial is ideal for any health professional or student interested in learning the fundamentals of electrocardiography. Internet access with either Microsoft Internet Explorer or Netscape Navigator browser software is required for log-on registration. A full screen monitor with 800×600 pixels resolution and a minimum of 256 colors imagery is recommended. Course registration includes 20 log-on sessions. Individuals intending to master ECGs through Internet-access education need to be goal oriented and self paced. It takes concentration and motivation to complete the logically sequenced package. Since Internet connections sometimes disconnect, participants may also need to be patient with disruptions in information transmission.

Study via the Internet can be a very rewarding experience for the self-directed person. The authors of this tutorial provide a wealth of information in a very charismatic fashion. The use of well-crafted graphics and vibrant colors add to the overall visual experience. The audience is treated to the latest in technology—including Java scripting and hypertext—that adds to the overall completeness of the study package. I found the glossary of terminology very useful. The course is peppered with humor that helps maintain the reader's interest and serves as a focal point for discussing the content. However, I did find the banter of students in the "cyber" classroom too flip at times.

Overall, I found this educational experience rewarding, and thought that the content was accurate, well organized, and on-topic. The appropriate repetition of important points, combined with exceptional graphics and animation, made the process exceptionally educational and reinforcing. The exams are well spaced and cover the material precisely, and the unit exams are a perfect review for the final exam.

I would recommend that the author consider adding digital still pictures or video of real patient applications that would help stimulate the thought process further. Scrolling through text and ECG strips alone can become tedious, despite the rich color palate. Actual lead placements or oscilloscope tracings would bring a stronger sense of realism to the already perfect ECG rhythm strips, and would be a natural extension of the content. Participating in this course reinforced my awareness of the increasing importance of technology in today's educational process, and highlighted the fact that Internet instruction can and should be more than reading text on a computer monitor. This course is a step in the right direction for medical education, and I look forward to other offerings from these authors. Kudos for their insight and ingenuity.

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Sterilizing Solution. Sporicidin International announces it has received Food and Drug Administration 510(k) clearance for Sporicidin Sterilizing Solution for use on critical and semi-critical medical devices including surgical instruments and equipment. According to the company, their product uses less than 1% glutaraldehyde, which they say is the lowest level in any such FDA-cleared product. Sporicidin says the product achieves a high level of disinfection in 20 minutes and that because of the reduced glutaraldehyde, Sporicidin Sterilizing Solution is safer for instruments and equipment. For more information from Sporicidin International, circle number 161 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Automated Sleep Scoring. Grass Instrument Division, Astro-Med Inc, announces a newly developed system for automated sleep staging. Grass has designated the new system CASS (Computer Assisted Sleep Staging) and says the system brings unsurpassed accuracy and speed to identifying the stages of sleep during an overnight recording of a patient. A company press release says the system analyzes very high frequency brain waves acquired with Grass amplifiers found within the PSG (polysomnograph). According to Grass, these high frequency brain waves have a direct correlation to a patient's stages of sleep, and the system identifies those stages of sleep as the overnight recording takes place. For more information from Astro-Med, circle number 162 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Infant Graphics Monitor. Mallinckrodt Inc has announced the availability of its new StarTrack™ Infant Graphics Monitor as an option for its Infant Star Ventilator line. A company spokesperson says that the new device allows physicians and therapists to obtain accurate readings of several parameters in time to establish appropriate initial settings and that the StarTrack measures expiratory tidal volume, minute volume, total respiratory rate, percent tube leak, and dynamic compliance. According to a company press release, the device displays two waveforms simultaneously and provides flow/volume and pressure/volume loops. For more information from Mallinckrodt Inc, circle number 163 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Patient Monitoring Software. Siemens unveils its newest version of Infinity™ patient monitoring software. A company press release says that a software upgrade for the product, version VE, supports added neurological monitoring capabilities and enhances the Infinity System's clinical utility for cardiac care, adult and neonatal ICU, and anesthesia. Siemens adds that the new software version also supports the new Infinity EEG Pod™ which allows Infinity monitors to carry a range of neurological parameters including EEG's symmetry, amplitude, and frequencies. Siemens say other added features include the ability to monitor the Frank-lead configuration, to calculate and store drug deliveries and titrations, and more. For more information from Siemens, circle number 164 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

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1. Patient identifier

In confidence

2. Age at time of event: or Date of birth:

3. Sex ☐ female ☐ male

4. Weight ____ lbs or ____ kgs

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)

☐ death ____ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other: ____

3. Date of event (mo/day/yr)

4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other: ____

5. Expiration date (mo/day/yr)

6.
model # ____
catalog # ____
serial # ____
lot # ____
other # ____

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address

phone #

2. Health professional? ☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

A Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for raducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H. Humphrey Building, Room 531-H
200 Independence Avenue, S.W.
Washington, DC 20201

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Please do NOT return this form to either of these addresses.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

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Public Health Service
Food and Drug Administration
Rockville, MD 20857

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MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name _____

First Name _____

Social Security No. _____

Home Address _____

City _____

State _____ Zip __ __ __ __ __ — __ __ __ __

Phone No. (_____) _____

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

Type of Business

- ☐ Hospital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify _____

Date of Birth (optional) _____ Sex (optional) _____

U.S. Citizen? ____ Yes ____ No

Have you ever been a member of the AARC? _____

If so, when? From _____ to _____

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Medical Director/Medical Sponsor _____

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify _____

Expected Date of Graduation *(REQUIRED INFORMATION)*

Month _____ Year _____

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature _____

Date _____

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associate (Foreign)	$102.50
☐ Special	$ 87.50
☐ Student	$ 45.00
TOTAL	$_____

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Cantinuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00
TOTAL	$_____

GRAND TOTAL = Membership Fee plus optional sections **$**_____

- ☐ Total Amount Enclosed/Charged $_____
- ☐ Please charge my dues (see below)

To charge your dues, complete the following:

- ☐ MasterCard
- ☐ Visa

Card Number

_ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _

Card Expires _____/_____

Signature _____

Mail application and appropriate fees to:

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • [972] 243-2272 • Fax [972] 484-2720

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to these Instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* [Respir Care 1997; 42(6):623-634]. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. To receive these Instructions and related materials, write to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104, call (206) 223-0558, or fax (206) 223-0563.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions. Comparative cost data should be included wherever possible.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References. Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication, marked "For publication," about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included.

Blood Gas Corner: A brief, instructive case report involving blood gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 Issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs; may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

Print on one side of white bond paper, 8.5 in. x 11 in. (216 x 279 mm) with margins of at least 1 in. (25 mm) on all sides of the page. Use ***double-spacing*** throughout the ***entire*** manuscript. Use a standard font (eg, Times, Helvetica, or Courier) at least 10 points in size, and

do not use italics except for special emphasis. Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. **Do *not* put authors' names, institutional affiliations or allusions to institutional affiliations in the text, or other identification anywhere except on the title page.** Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

References. Cite only published works as references. Manuscripts accepted but not yet published may be cited as references: designate the accepting journal, followed by (in press), and provide 3 copies of the in-press article for reviewer inspection. Cite references in the text with superscript numerals. Assign numbers in the order that references are first cited. On the reference page, list the cited works in numerical order. Follow the Journal's style for references. Abbreviate journal names as in *Index Medicus*. List all authors.

Article in a journal carrying pagination throughout volume:

> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

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Commercial Products. In parentheses in the text, identify any commercial product (including model number if applicable) the first time it is mentioned, giving the manufacturer's name, city, and state or country. If four or more products are mentioned, do not list any manufacturers in the text; instead, list them on a Product Sources page at the end of the text, before the References. Provide model numbers when available and manufacturer's suggested price, if the study has cost implications.

Ethics. When reporting experiments on human subjects, indicate that procedures were conducted in accordance with the ethical standards of the *World Medical Association Declaration of Helsinki* [Respir Care 1997;42(6):635-636] or of the institution's committee

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Statistics. Identify the statistical tests used in analyzing the data, and give the prospectively determined level of significance in the Methods section. Report actual p values in Results. Cite only textbook and published article references to support choices of tests. Identify any general-use or commercial computer programs used, naming manufacturers and their locations. These should be listed on the product-sources page.

Units of Measurement. Express measurements of length, height, weight, and volume in metric units appropriately abbreviated; temperatures in degrees Celsius; and blood pressures in millimeters of mercury (mm Hg). Report hematologic and clinical-chemistry measurements in conventional metric and in SI (*Systémé Internationale*) units. Show gas pressures (including blood gas tensions) in torr. List SI equivalent values, when possible, in brackets following non-SI values—for example, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33(10):861-873 (Oct 1988), 1989;34(2):145 (Feb 1989), and 1997;42(6):639-640 (June 1997).

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Please use the following forms: cm H_2O (not cmH2O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), mL (not ml), mm Hg (not mmHg), pH (not Ph or PH), $p > 0.001$ (not p>0.001), s (not sec), S_{pO_2} (pulse-oximetry saturation). See RESPIRATORY CARE: Standard Abbreviations and Symbols [Respir Care 1997;42(6):637-642].

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Mail three copies [1 copy with author(s) name(s), affiliation(s), 2 copies without name(s) and affiliation(s) for reviewers] of the manuscript, figures, and 1 diskette, and the Cover Letter & Checklist to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Do not fax manuscripts. Protect figures with cardboard. Keep a copy of the manuscript and figures. Receipt of your manuscript will be acknowledged.

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Author Signature/Date _____

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Calendar of Events

AARC & AFFILIATES

September 8-10—*Hot Springs, Arkansas*

The ASRC presents its 28th Annual State Meeting and Education Seminar at the Hot Springs Hilton and Convention Center. Topics will include health care in Arkansas, patient rights, disease management, and case studies by area RTs. Scheduled to speak are State Representative Brenda Gullett, Congressman Vic Snyder, Tom Kallstrom of Cleveland, OH, ASRC President John Campbell, AARC President Dianne Kimball, and Kristin McFall of Denver, CO (topic: cystic fibrosis).
Contact: John Lindsey at (870) 541-7606, jlindsey@ahecpb.uams.edu

September 16–17—*Pittsburgh, Pennsylvania*

The PSRC will host their 26th Annual Western Pennsylvania Regional Pulmonary Medicine and Physiology Conference at the Sheraton Station Square. Topics include management, critical care, sleep diagnostics, pulmonary rehabilitation, and the physician forum.
Contact: Debbie Logan at (800) 545-4663, ext. 112.

September 24–25—*Cleveland, Ohio*

The AARC presents the "Disease Management of Asthma" seminar. Come and join a distinguished faculty as they review the NIH asthma guidelines, marketing the asthma program, pharmacology, and numerous other aspects of asthma program management.
Contact: The AARC Conventions Office at (972) 243-2272.

October 1—*Melville, New York*

The NYSSRC's Southeastern Chapter hosts their 31st annual symposium, "Respiratory Care — A Work in Progress," at the Huntington Hilton Hotel in Melville, Long Island. The keynote address will be given by Carl Wiezalis, vice-president of the AARC.
Contact: For information, call Jim Ganetis at (516) 444-3181 or **www.nyssrc.org**.

October 20–22—*Daniels, West Virginia*

The West Virginia Society for Respiratory Care will host its Annual Fall Meeting at the Glade Springs Resort, Country Inns and Suites.
Contact: For more information, contact Jay Wildt, co-chair of program and education, at (304) 442-7474.

December 13–16—*Las Vegas, Nevada*

The AARC's 45th International Respiratory Congress is scheduled for Dec. 13–16 (Monday through Thursday) at the Las Vegas Convention Center. Sessions appealing to all levels of health care providers will be offered, with CRCE credit available. Exhibits by international manufacturers of cardiopulmonary equipment will be featured. Additional information will be available in the fall.

Other Meetings

September 16-19—*Phoenix, Arizona*

The American Association of Cardiovascular and Pulmonary Rehabilitation will hold its 14th annual meeting at the Phoenix Civic Center.
Contact: For more information, call (608) 831-6989; aacvpr@tmahq.com; http://www.aacvpr.org.

October 1–3—*Ottawa, Ontario, Canada*

The Canadian COPD Alliance will host "Building and Enriching Partnerships in the Management of COPD" at the Radisson Hotel Ottawa Centre. This conference will include plenary sessions on the epidemiology of COPD and scientific workshops on spirometry, smoking cessation, and the evidence to support management approaches to COPD. Included is a series of practical workshops on rehabilitation and a parallel consumer track for those who live with COPD.
Contact: call (613) 747-6776 or see their web site at **www.lung.ca/CCA/conference**.

October 4–5—*Ann Arbor, Michigan*

The Office of Continuing Medical Education at the University of Michigan is sponsoring a conference, "Update on Pulmonary and Critical Care Medicine," at the Towsley Center.
Contact: For more information, contact Laura Castellanos at (734) 647-8784.

October 31–November 4—*Chicago, Illinois*

The American College of Chest Physicians will host their 65th Annual International Scientific Assembly at the Lakeside Center. For information, contact Member Services at (800) 343-2227, fax (847) 498-5460, or **www.chestnet.org**.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
- —Current job listings
- —American Respiratory Care Foundation fellowships, grants, & awards
- —Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
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Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

RC Week Online
http://www.aarc.org/rcweek/

The National Board for Respiratory Care—1999 Examination Dates and Fees

Examination	Examination Date	Examination Fee
CRTT (CRT) Examination	November 13, 1999 Application Deadline: September 1, 1999	$120 (new applicant) 80 (reapplicant)

Information on the 2000 Examination Fees is not available.

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156,or e-mail: nbrc-info@nbrc.org

WATCH FOR

OPEN FORUM ABSTRACTS

OCTOBER 1999

Asthma Disease Management Course Comes to Cleveland

"Disease Management of Asthma," a newly designed day-and-a-half course, will be held in Cleveland Sept 24-25. Drawing on the expertise of a nationally recognized faculty, this course has been developed in accordance with the National Institute of Health *Asthma Guidelines for the Diagnosis and Management of Asthma*. Completing this course will earn respiratory therapists 11 hours of continuing education credit. Topics covered include disease management, pharmacology, educational strategies, marketing your program, outcomes management, and age-specific issues. Register on-line or call the AARC for registration materials. The program will be held September 24-25, at the Marriott Cleveland Airport Hotel. Registration before August 30, 1999 is $175 for AARC members, $250 for non-members. (After August 30, register for $275 member, $350 non-member)

AARC Exhibition Becomes 'Selling Show'

The AARC's 45th International Respiratory Congress will, for the first time in its history, provide attendees and exhibitors the opportunity to negotiate sales of products and equipment at the exhibitor show, December 13 -16, in Las Vegas. More than 200 equipment manufacturers, pharmaceutical companies, service organizations, and supply companies will exhibit at the show. "We've always had an extremely enthusiastic response to our exhibiting companies," said AARC Executive Director Sam Giordano. "We decided to take that approach this year to make attending the meeting even more cost effective — and the show specials and discounts our exhibitors are providing will make the exhibition an even greater success," he said.

New Products Help with Clinical Practice

These products from the AARC, some newly introduced, will help in both clinical practice situations and in administration, in acute care and post-acute care. Add $8.25 to quoted price for shipping and handling. Texas residents add sales tax.

Diagnostic Training and Competence Assessment Manual for Pulmonary and Noninvasive Cardiology. CDRom. $267 for AARC members. This manual (on compact disk) is ideal for use in course development, training, orientation, and competence assessment for individuals in the laboratory setting. A pulmonary diagnostics section features quality control, diffusing capacity, whole body plethysmography, arterial blood gas sampling, bronchoscopy and seven other procedures. The noninvasive cardiology section features EEG, stress testing, event monitoring, exercise testing and three other procedures.

Asthma Disease State Management. Video and workbook. $79.95 for members. Provides instruction in how to create an effective asthma disease management program in your facility. Covers diagnosis, pharmacological therapy, environmental controls, patient/family education, and case studies. Approved for two hours of CRCE credit and nursing CE credit.

Orientation and Competency Assurance Documentation Manual. Manual. $65 for members. Provides the information, assessment tools, and models necessary to demonstrate that the competence of employees is documented according to JCAHO requirements.

Uniform Reporting Manual for Acute Care. Manual. $65 for members. Provides you with nationally recognized standards for documenting workload units and time standards. Includes patient assessment activities and covers bronchial hygiene, supplemental oxygen, airway care, diagnostic tests, and cardiovascular diagnostics.

Uniform Reporting Manual for Subacute Care. Manual. $75 for members. Provides tools to determine productivity, track trends in the utilization of respiratory care services, assist in determining personnel requirements, measure demand for and intensity of services, and meet the requirements of prospective payment systems (PPS).

Respiratory Home Care Procedure Manual. Manual. $80 for members. The new "Respiratory Home Care Procedure Manual" is specifically designed for the home care setting. And, it is easily adaptable to any alternate care site from subacute to home medical equipment companies and nursing agencies. The manual features five sections of information, forms, and checklists for the patient and practitioner.

Authors in This Issue

Axton Jr, Kenneth L 1139
Cascade, Philip N 1033
Collins, Jannette 1044
Doorley, Patricia A 1015
Durbin Jr, Charles G 1015
Gross, George W 1095
Henry, Daniel A 1064
Kazerooni, Ella A 1033
Klein, Jeffrey S 1078
MacMahon, Heber 1018
Miller Jr, Wallace T 1127
Morton, Douglas W 1137
Moufarrej, Ramzi 1137
Nanavaty, Uday 1138
Stern, Eric J 1017
Tarver, Robert D 1017

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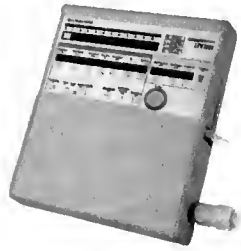

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Collins, Jannette 1044
Doorley, Patricia A 1015
Durbin Jr, Charles G 1015
Gross, George W 1095
Henry, Daniel A 1064
Kazerooni, Ella A 1033
Klein, Jeffrey S 1078
MacMahon, Heber 1018
Miller Jr, Wallace T 1127
Morton, Douglas W 1137
Moufarrej, Ramzi 1137
Nanavaty, Uday 1138
Stern, Eric J 1017
Tarver, Robert D 1017

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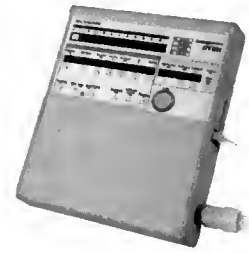

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1 McIlwaine, P.M., Wong, L.T., Peacock, D., Davidson, G.F. "Long-term comparative trial of conventional postural drainage and percussion versus positive expiratory pressure physiotherapy in the treatment of cystic fibrosis." *The Journal of Pediatrics*, October 1997.

2 Protocol of the Month, University of Pittsburgh Medical Center, Department of Respiratory Care. *AARC Times*, May 1997.

3 Mahlmeister MJ, Fink JB, Hoffman GL, Fifer LF, "Positive-expiratory-pressure mask therapy: Theoretical and Practical Considerations and a Review of the Literature", *Respiratory Care*, 1991; 36:1218-1230.

4 "AARC Clinical Practice Guideline: 'Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy'," *Respiratory Care*, 1993; 38:516-521.

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